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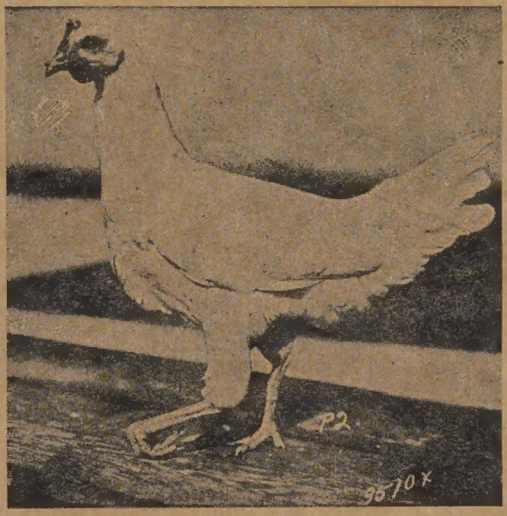
UNIVERSITY OF ALBERTA
FACULTY OF AGRICULTURE

DISEASES OF POULTRY

BY

D. R. CLANDININ
Department of Animal Science



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Diseases of Poultry

BY

D. R. CLANDININ

Assistant Professor of Poultry Husbandry

Introduction

This circular, originally published under the authorship of Miss Helen I. Milne, has been revised and extended in response to many requests from poultry keepers for information regarding poultry diseases. In the discussion, an effort is made, first, to outline practices applicable to the prevention of disease in poultry, and second, to deal with the more important diseases to which poultry are susceptible.

The writer desires to acknowledge his indebtedness to Professors E. A. Lloyd and J. Biely of the University of British Columbia, and to Dr. T. L. Jones and C. W. Traves of the Provincial Department of Agriculture, and to W. Roach of the Dominion Department of Agriculture for critically examining the manuscript. He is indebted to Drs. T. W. M. Cameron and W. E. Swales of the Institute of Parasitology, Macdonald College, for critically examining the sections on internal and external parasites.

Prevention of Disease

Domestic poultry are subject to a large number and variety of diseases, some of them highly contagious, many of them incurable, and all of them a source of loss to the poultryman. While treatment may be applied successfully in a limited number of cases, it should not be considered the solution to the disease problem. The most economical and satisfactory line of attack in combating disease lies in prevention. This involves the intelligent application of the principles of breeding, feeding and sanitation, combined with prompt recognition of abnormal conditions, as they occur, and careful, thorough and efficient treatment at the first sign of trouble.

Breeding.

Of fundamental importance to the well-being of the flock are the inherited qualities of the birds in it. For this reason, the culling, selection and breeding in the flock should be such that constitutional vigour and resistance to disease will be developed. From the time chicks hatch, a rigid and continuous culling of weaklings should be practised. Any birds that appear to be lacking in vigour, are unthrifty or diseased, should be culled out and disposed of as soon as noticed. Only vigorous, healthy two-year,

or older birds should be used as breeders. Pullets should not be used as breeeders, unless they show exceptionally good performance and vigour.

When it is found necessary to introduce new blood into a flock, care should be taken to see that the imported stock comes from sources known to be high in stamina and free from disease. This is advisable, first, because resistance or susceptibility to disease may be transmitted and, second, because many poultry diseases can be introduced into a flock by "carrier" birds.

Feeding.

Good feeding has also been shown to play an important rôle in resistance to disease. Over a period of years, the approximate protein, carbohydrate, fat, fibre, mineral and vitamin requirements of various classes of poultry have been determined. Accordingly, lowered resistance to, and mortality from disease due to dietary deficiencies need no longer occur, even where birds are kept in strict confinement. The inclusion of these nutrients in the diet in proper amounts and proportions will be found of inestimable value in safe-guarding the health of the flock.

Sanitation.

Agents responsible for disease may be introduced into a flock by means of new birds brought in from other flocks; by birds returned from poultry shows; by contamination from feed sacks or shipping crates; by the feet of humans; by wild birds and animals; and by parasites (such as mites). It is wise, therefore, to take precautions against such possible sources of infection by purchasing new stock only from flocks known to be free from disease; by isolating newly purchased stock and birds returned from poultry shows for two to three weeks before placing them in with the rest of the flock; by excluding poultry buyers and visitors from the poultry yards and houses; and by providing every possible means of guarding the flock against infection from outside sources.

On the farm, an intelligent and conscientious program of sanitation should be carried out so that the birds may live in comfortable, clean and uncontaminated surroundings, and so that in case of an outbreak of disease, its spread can be checked quickly.

The laying house should be located on well-drained land, face south or south-east and should be protected by a windbreak. It should be of ample size for the number of birds being kept, properly insulated against extremes of temperature, well-ventilated but free from drafts, dry and sunny. The walls of the house should be of tight boarding, the floor of cement or good lumber. The inside equipment should include: removable dropping boards, roosts, broody coop, nests, mash hoppers and waterers on stands, grain troughs, and oyster shell and grit feeders.

Prior to placing each year's pullets in the laying house for the winter, it should be given a thorough cleaning. Roosts, dropping boards, nests and other equipment should be removed for this purpose. These, as well as the floor of the house, should be scraped, swept, washed with water, scrubbed with lye-water (1 can of lye to 5 gallons of water), allowed to soak overnight, washed again with water and then disinfected by spraying with a good disinfectant (Iodine Suspensoid or Izal). If mites have been a source of trouble during the previous year, the house and equipment should also be treated as recommended under Mites (see Common Chicken Mite). Following cleaning and disinfecting, the equipment and walls of the house should be given a good coat of whitewash. Whitewash may be prepared by slaking one bushel of quickline in twelve gallons of hot water, dissolving two pounds of common salt and one pound of zinc-sulphate in two gallons of boiling water, pouring this mixture into the slaked lime, adding two gallons of skim milk and mixing thoroughly.

The floor of the laying house should be kept well covered with clean, dry litter. As a general rule the litter should be removed at weekly intervals during the winter, while removal every two to four weeks will usually suffice in summer. Nests should be kept well furnished with clean straw. Replenishing nesting material as required and cleaning nests at two-week intervals is advisable. Dropping boards should be kept clean; daily cleaning is recommended. All material removed from the house should be disposed of in such a manner that the birds will not have access to it.

In the daily routine nothing is more important than the providing of clean, fresh water in clean vessels, so designed and placed that they cannot readily become polluted, and the feeding of a good ration in clean hoppers and troughs. Water troughs and mash hoppers should be equipped with reels and stands. Scratch grain should be fed in long V-shaped troughs; litter feeding of scratch is insanitary. Grain troughs should be hung up when not in use so as to prevent unnecessary soilage.

The colony brooder house, like the laying house, should be of ample size for the number of birds for which it is intended, properly insulated if intended for early brooding, well ventilated but free from drafts, and dry. The house should have a double-boarded floor to ensure floor warmth and freedom from floor drafts, and should be built on a pair of good skids to facilitate moving. The house equipment should include: chick guard, mash and grain troughs of various sizes, waterers on wire stands, and forcing roosts that are wired underneath to prevent the chicks from coming into contact with their droppings.

The brooder house should be given a thorough cleaning (similar to that outlined for fall cleaning of a laying house) in the fall after the young stock has been transferred to winter quarters. Following cleaning, whitewashing and disinfection, the brooder house

should be moved into position on the piece of ground that is to be used for range the next season.

The floor of the brooder house should be kept well covered with clean, dry litter. During the first two weeks of brooding, the litter (especially under the hover) should be removed daily. Later weekly cleaning will suffice, and, as the birds get to staying outside most of the day, cleaning every two weeks will be found adequate.

As with layers, nothing is more important than the providing of clean, fresh water in clean vessels, and the feeding of a good ration in clean hoppers and troughs. Mash troughs for young chicks should be equipped with reels and stands in order to prevent feed soilage. Range hoppers for older chicks should be weatherproof and should be moved a short distance at each filling so as to avoid excessive contamination of the ground in one place. Scratch grain should be fed in long V-shaped troughs for the first ten weeks, after which it can be better supplied in open range hoppers. Water vessels should be provided with low wired-in stands so as to prevent the chicks from coming into contact with the damp, dropping-polluted, germ-infested litter or ground usually present about water containers.

The ground over which poultry range should be kept clean. This is necessary since many disease producing germs and parasites to which poultry are subject are soil borne. The easiest way to ensure clean ground for poultry is to follow a system of range rotation. A two-year rotation is recommended for adult stock; a three-year rotation for young stock. In the former, the ground about the laying house is so fenced as to provide for two yards of approximately equal size. These yards are used in alternate years. The yard not occupied in any one year is cleaned up by frequent cultivation or by cultivating for garden use. In the three-year rotation for young stock, three strips of range are required. Each piece of ground is used one year and rested two years. During the first year of rest the ground is cultivated frequently. Alfalfa or other range is sown in the spring of the second year of rest. The strips of ground do not have to be fenced but should be far enough apart to prevent the birds from running over the previous year's range.

Besides using a system of range rotation there are a number of other precautions that should be taken in order to ensure clean ground for poultry. Firstly, adult chickens should not be permitted access to the young stock ranges. Adult stock are carriers of disease germs and would contaminate the young stock ranges. Secondly, chickens should not be allowed access to turkey ranges and vice versa. This is necessary in order to control blackhead and gapeworms (see Blackhead and Gapeworm). Thirdly, every effort should be made to keep the ranges clean and sanitary; low, wet spots should be filled in or fenced off so as to lessen chances of

parasitic and coccidial infestation (see Internal Parasites and Coccidiosis); the ground about the brooder house should be swept each time the house is cleaned and the brooder house should be moved a short distance every four weeks to avoid excessive contamination of the ground in one place.

Under conditions where rotation of ranges is not possible, the use of wire floored sun-porches, or small fenced yards kept clean by changing the top three or four inches of soil yearly and cultivating frequently, is recommended. It is much better to confine poultry to a small piece of clean ground than to let poultry range at large over dirty ground.

Diagnosing Common Ailments.

Most of the ailments to which poultry are susceptible can be readily recognized on case history, symptoms, and post-mortem findings.

Case history often proves of value in ascertaining the cause of the trouble. A knowledge of such factors as the ration fed, feeding routine and whether or not the birds are confined or are on good range, frequently permits one to arrive at a more accurate diagnosis than one otherwise could make on symptoms and post-mortem findings alone. It often offers a means of confirming one's diagnosis and for this reason should not be overlooked.

When endeavouring to determine what is wrong with a bird, first take note of the symptoms displayed. Next, made a systematic examination of the bird for outward evidences of the presence of disease. Then make a similar examination of the bird for internal evidence of disease. Finally, diagnose the trouble in the light of the case history. Tables I and II will be found of assistance in arriving at a diagnosis. The reader is referred to the pages that follow for details on the various diseases.

TABLE I.
EXTERNAL AND INTERNAL SYMPTOMS IN YOUNG BIRDS

DISEASE	SYMPTOMS	
	External	Internal
Fowl Pox	Wart-like lesions about head, canker-like patches in mouth and throat.	None.
Infectious Bronchitis	Watery eyes, mucus about nostrils, sneezing, labored breathing, wheezing, coughing, choking, high mortality.	Purulent exudate in trachea (windpipe), congested lungs.
Fowl Leukosis	Paralysis of legs, wings and neck; depigmentation of iris of eye, derangement of pupil of eye and blindness; paleness about head.	Swollen and discolord sciatic (leg) and brachial (wing) plexuses (nerves), tumor-like masses in visceral organs, watery blood, enlarged liver, spleen and kidneys.
Pullorum	Droopiness, plaintive chirping, diarrhoea with pasting up behind, high mortality.	Nodules in heart and lungs, unabsorbed yolk.

TABLE I—Continued

DISEASE	SYMPTOMS	
	External	Internal
Coryza	Watery eyes, sneezing, serous or thick foul-smelling discharge from nostrils, swollen sinuses, cheesy material in mouth cleft, labored breathing, wheezing.	None.
Botulism	Sleepiness, limpness of neck and wings, unsteady gait, prostration, looseness of feathers.	None.
Omphalitis	Pus about navel, puffy abdomen, diarrhoea.	Putrefaction about navel, unabsorbed yolk, distended gall bladder.
Coccidiosis	Loss of appetite, emaciation, droopiness, passage of blood in droppings. Also as in Table II.	Enlarged caeca (blind pouches of intestine), thickening and inflammation of walls of caeca, blood or blood-stained material in caeca. Also as in Table II.
Blackhead	Loss of appetite, emaciation, poor growth, increased thirst, droopiness, orange brown or green diarrhoea.	Ulcer-like areas of pink or yellowish color in liver, enlarged caeca, thickening and ulceration of walls of caeca.
Aspergillosis	Loss of appetite, emaciation, sneezing, wheezing, gasping for breath.	Grayish or greenish lesions in trachea, bronchi, lungs and air sacs.
Thrush	Loss of appetite, emaciation, labored breathing, ulceration of vent.	Cankerous growths in mouth, ulceration of gullet, crop, proventriculus, gizzard and intestine.
Nutritional Roup	Loss of appetite, poor growth, staggering gait, soreness of eyes, swollen sinuses, drowsiness, diarrhoea.	White deposits in kidneys and cloaca (chamber just inside vent) and on internal organs.
Curled-toe Paralysis	Paralysis of legs, inward curling of toes.	None.
Rickets	Staggering gait, reluctance to stand or move, enlarged joints, bent legs, soft beaks, crooked breast bones, poor growth, general unthrift.	Beaded ribs, piling up of cartilage at ends of long bones.
Perosis	Bowing or twisting of legs, slipping of Achilles' tendon (large tendon at hock joint) from its normal position, enlargement and flattening of hock joint.	None.
Gout	Dark comb, unsteady gait, lameness, swollen joints.	Enlarged kidneys, white deposits on internal organs.
Beak Necrosis	Accumulation of mash around mouth, deformed beak.	None.
Impaction of the Crop	Loss of appetite, general distress, crop distended and doughy to feel.	Fibrous material in crop.
Pendulous Crop	Enlarged baggy crop.	None.
Impaction of the Gizzard	Loss of appetite, general unthrift.	Fibrous material in gizzard.
Bumblefoot	Swollen foot.	None.
Cannibalism	Toes or tail bleeding, feathers missing on back and abdomen, vent bleeding.	None.
Lice	Straw colored elongated insects found on bird at any time.	None.

TABLE 1—Continued

DISEASE	SYMPTOMS	
	External	Internal
Common Chicken Mite	Gray or red ovoid insects found on bird at night.	None.
Northern Fowl Mite	Gray or red ovoid insects found on bird in localized areas at any time.	None.
Depluming Mite	Feathers absent in certain areas.	None.
Large Intestinal Roundworm	Paleness of head and shanks, loss of appetite, poor growth, emaciation, droopiness, paralysis.	Long, white, unsegmented worms in intestine.
Caecal Worm	As in Large Intestinal Roundworm.	Small, white unsegmented worms in caeca.
Gapeworm	Inactivity, gasping for breath, death by suffocation.	Y-shaped worms in trachea.
Tapeworm	As in Large Intestinal Roundworm.	Long or short segmented worms in intestine.

TABLE II.

EXTERNAL AND INTERNAL SYMPTOMS IN ADULT BIRDS

DISEASE	SYMPTOMS	
	External	Internal
Fowl Pox	As in Table I.	As in Table I.
Infectious Laryngo-tracheitis	Watery eyes, mucus about nostrils, sneezing, labored breathing, wheezing, coughing, choking, high mortality.	Blood-stained exudate in trachea (windpipe), congested lungs.
Infectious Bronchitis	As in Table I.	Catarrhal exudate in trachea, congested lungs.
Fowl Leukosis	As in Table I.	As in Table I.
Pullorum	None.	Misshapen, greenish or leaden colored yolks in ovary, enlarged and noduled heart, pericarditis (inflammation of the heart sac).
Fowl Tuberculosis	Extreme loss of weight, lameness, swollen joints, diarrhoea, paleness about head.	Grayish or yellowish raised tubercles of cheesy consistency in liver and spleen and on intestines.
Fowl Typhoid	Droopiness, sleepiness, paleness about head, greenish or yellowish diarrhoea, high fever, increased thirst.	Greenish bronze liver, enlarged spleen and kidneys, misshapen greenish or leaden colored yolks in ovary, enlarged and noduled heart, pericarditis.
Fowl Cholera	Sudden death, loss of appetite, droopiness, sleepiness, darkness about head, greenish or yellowish diarrhoea, high fever, increased thirst, restrained breathing.	Sticky mucus in mouth, congestion of visceral organs, friable liver, pinhead sized necrotic areas in liver, small haemorrhages on internal organs.
Coryza	As in Table I.	As in Table I.

TABLE 2—Continued

DISEASE	SYMPTOMS	
	External	Internal
Botulism	As in Table I.	As in Table I.
Coccidiosis	Loss of appetite, progressive emaciation, sluggishness, paleness about head, paralysis of legs. Also as in Table I.	Inflammation and thickening of walls of upper intestine. Also as in Table I.
Blackhead	Progressive emaciation, sunken eyes, sluggishness, diarrhoea.	As in Table I.
Aspergillosis	As in Table I.	As in Table I.
Favus	Dirty white or yellowish scaly lesions about head.	None.
Thrush	As in Table I.	As in Table I.
Nutritional Roup	Low hatchability, decreased egg production, loss of appetite, incoordinated gait, soreness of eyes, swollen sinuses, droopiness.	White deposits in kidneys and cloaca and on internal organs, white pustules in mouth, pharynx and gullet.
Riboflavin Deficiency	Low hatchability, decreased egg production.	None.
Rickets	Low hatchability, decreased egg production, production of thin shelled abnormally small eggs, weak and crooked bones.	None.
Gout	As in Table I.	As in Table I.
Obesity	Sluggishness.	Excess of abdominal fat.
Beak Canker	Sore at beak angle.	None.
Impaction of the Crop	As in Table I.	As in Table I.
Pendulous Crop	As in Table I.	As in Table I.
Impaction of the Gizzard	As in Table I.	As in Table I.
Bumblefoot	As in Table I.	As in Table I.
Ascites	Distended abdomen.	Serous fluid in body cavity.
Ruptured Yolk	Dark comb, sagging of abdomen.	Yolk material in body cavity.
Intra-ovulation	Dark comb, sagging of abdomen.	Yolks in body cavity.
Impacted Oviduct	None.	Hardened yolk and albumen in oviduct (egg tube).
Ruptured Oviduct	None.	Break in wall of oviduct.
Internal Laying	Sagging of abdomen.	Eggs and egg material in body cavity.
Egg Bound	Straining to lay.	Egg in lower part of oviduct.
Prolapsus	Eversion of cloaca (chamber just inside vent) and lower oviduct.	None.
Cloacitis	Swelling, inflammation and ulceration of vent, obnoxious odour.	Inflammation and ulceration of cloaca.
Cannibalism	As in Table I.	As in Table I.
Lice	As in Table I.	As in Table I.

TABLE II—Continued

DISEASE	SYMPTOMS	
	External	Internal
Common Chicken Mite	As in Table I.	As in Table I.
Northern Fowl Mite	As in Table I.	As in Table I.
Scaly Leg Mite	Scales on legs raised.	Ovoid insects under scales.
Depluming Mite	As in Table I.	As in Table I.
Large Intestinal Roundworm	Decreased egg production, loss of weight, unthrift.	As in Table I.
Caecal Worm	As in Large Intestinal Roundworm.	As in Table I.
Gapeworm	As in Table I.	As in Table I.
Tapeworm	As in Large Intestinal Roundworm.	As in Table I.

VIRUS DISEASES

A number of the more troublesome diseases to which poultry are susceptible are caused by filterable viruses. (Filterable viruses are disease-producing agents of ultra-microscopic size.) Fowl pox, infectious laryngotracheitis, infectious bronchitis, and fowl leukosis are among the more important diseases in this group.

Fowl Pox.

Fowl pox, also known as chicken pox and avian diphtheria, is a highly infectious disease, to which both chickens and turkeys are susceptible. This disease is encountered most frequently during the fall and winter months, and is more prevalent at the Pacific coast than it is in the prairie and eastern provinces of Canada.

Fowl pox may manifest itself in one or both of two forms—the skin form and the diphtheritic form. The skin form is characterized by brownish wart-like lesions on the comb, face, wattles, earlobes and eyelids (see Fig. 1). The diphtheritic form is characterized by canker-like patches in the mouth and throat. Both forms may be accompanied by loss of appetite, loss of weight and de-

Fig. 1—Wart-like lesions on comb, face, wattle, and earlobe of a fowl affected with fowl pox. (After California Agricultural Experiment Station.)

creased egg production. Post-mortem examination reveals no internal changes. Diagnosis is usually made on the basis of pox lesions and the infectious nature of the malady, though inoculation experiments offer the only means of proving the presence of this disease.

Feed and water constitute the principal agents responsible for the spread of fowl pox in a flock. The virus causing the disease gets into the feed and water while affected birds feed and drink. Later, it gains entrance to the bodies of healthy birds through skin and mouth abrasions.

Mortality from this disease varies greatly, depending on the severity of the outbreak. In most outbreaks, mortality losses are light, but financial losses through delayed egg production in pullets and decreased egg production in laying hens are great.

Where an outbreak of fowl pox has occurred, the following control measures are recommended:

(1) Remove and isolate all affected birds.

(2) Thoroughly clean and disinfect the houses and equipment.

(3) Vaccinate apparently healthy birds. If it is a flock of vigorous growing birds that is affected, use chicken pox vaccine for vaccination. It induces solid immunity. If it is a flock of none-too-vigorous growing birds or a flock of laying birds, use pigeon pox vaccine. It is less severe than chicken pox vaccine. Pigeon pox vaccine induces only temporary immunity.

(4) Remove and isolate any other birds showing evidence of infection.

Many of the birds removed from the flock may be saved by proper treatment. Treatment entails removing the wart-like lesions and cankerous growths and painting the raw areas with 5% iodine, 10-15% argyrol or 4% silver nitrate solution. The wart-like lesions should be softened with warm salt and water before removal. Birds that survive an attack of fowl pox are in most cases immune to further infection from this disease.

Once fowl pox has appeared on a farm, yearly vaccination of young stock with chicken pox vaccine should be practised. The best time to vaccinate growing chickens and growing turkeys is when they are eight to fourteen and ten to twelve weeks old respectively. Under no circumstances should unthrifty stock be vaccinated. If such is done, in all probability an outbreak of the disease will occur.

Two methods are commonly employed in connection with vaccination for fowl pox, namely, the follicle and stick methods. The follicle method is used with pigeon pox vaccine on laying and none-too-vigorous growing birds. It consists of removing six or eight feathers in the upper thigh region and brushing into the follicles a small amount of a water suspension of the virus. The stick method, on the other hand, is used with chicken pox vaccine

on vigorous growing chickens and on all classes of turkeys. The method consists of piercing the wing web with the eye ends of two fine needles which have been previously dipped in vaccine. To carry out this method, there is needed a three by one half-inch tube provided with a rubber stopper in which are inserted, with eye ends out, two fine needles. With both methods of vaccination "takes" are read 7-10 days after vaccination. Birds that do not show takes should be revaccinated. Immunity develops in four weeks.

Vaccination of young stock in flocks where fowl pox has not as yet occurred, should not be practised.

Infectious Laryngotracheitis.

This is an acute, very infectious disease to which chickens are highly susceptible. Generally speaking, the disease is more common in adult chickens than in young chickens. Outbreaks of laryngotracheitis occur mainly during the fall and winter months. This disease is more prevalent in the prairie and eastern provinces of Canada than it is at the Pacific coast.

The first symptoms of this dease are watery eyes and sneezing. As the disease progresses, breathing becomes more difficult and wheezing and rattling sounds are audible. During coughing spells that follow, afflicted birds shake their heads in an effort to dislodge mucous exudate that accumulates in the larynx and trachea (windpipe). In advanced stages, affected birds take on a sitting posture extending their heads during each inspiration of air (see Fig. 2). Mortality is usually highest on the third or fourth day after exposure to the disease. The disease runs its course in from 7-15 days, though symptoms may be manifested for longer periods than this. Birds that recover from the disease harbour the causative virus in their throats for months, and thus serve as a source of infection to healthy birds.

Fig. 2.—Typical posture of fowl during inspiration in advanced stage of laryngotracheitis. (After California Agricultural Experiment Station.)

Post-mortem examination shows mucus about the nostrils and the presence of bloody exudate in the larynx and trachea. Small haemorrhages are also seen in the larynx and trachea. In some instances, a yellowish cheesy exudate may be found in the larynx and trachea. Diagnosis of

laryngotracheitis is usually made on the aforementioned symptoms and post-mortem findings and on the rapidity of spread of the infection. Positive proof of the presence of laryngotracheitis infection, however, can only be obtained by carrying out inoculation experiments.

Larnygotracheitis may be introduced into a flock through the purchase of carrier birds, or by bringing contaminated shipping crates, feed sacks, etc., on the premises. The disease is spread from one bird to another through the air.

Laryngotracheitis is considered to be one of the most serious diseases with which poultrymen have to contend. Very heavy losses, both in birds and egg production, are often sustained when this malady appears.

Success in getting an outbreak of laryngotracheitis under control depends to a considerable extent on the stage at which the disease has reached when detected. At the first sign of this trouble, all sick birds should be removed from the flock and the houses and equipment should be thoroughly cleaned and disinfected. The virus is readily destroyed by 1% lye, 3% cresol or 5% carbolic acid solution. Further cases should be watched for and removed from the flock as soon as noticed. Birds removed from the flock should be disposed of immediately by burning. It is useless to treat affected birds, for even if treatment should prove successful, the recovered birds cannot be re-admitted to the flock for they retain the causative virus in their throats long after recovery. In flocks where the disease has a good start, all birds that have been exposed to the disease should be disposed of, and the houses and equipment should be thoroughly cleaned and disinfected. No poultry should be housed in the depopulated buildings until at least three months time has elapsed.

Since laryngotracheitis is introduced into a flock by mechanical means, i.e., on the shoes or clothes of visitors, on returned shipping crates, on feed sacks, etc., or through the importation of diseased or "carrier" stock, measures of prevention obviously must entail consideration of these factors. Visitors who have recently had direct contact with this disease should not be allowed on the premises. Others should be required to disinfect their shoes before being permitted to enter the yards and houses. The handling of birds by visitors should be discouraged. Shipping crates should be washed and disinfected immediately on arriving back on the premises. Feed sacks should be thoroughly disinfected before being used for hauling feed about the farm. New stock should only be purchased from flocks that have never experienced this disease.

In recent years, vaccination against laryngotracheitis has been introduced. In vaccinating for laryngotracheitis, the vaccine is applied to the mucous membranes of the cloaca and bursa through the vent by means of a swab. Takes are read four or five days

after vaccination. Birds that do not show takes are revaccinated. Immunity develops in 4-9 days. Best results are obtained when birds are vaccinated at 2-4 months of age.

It should be pointed out that in flocks where laryngotracheitis has never occurred and in flocks where the disease has occurred and where it has been brought under control by depopulation, vaccination should not be practised. Vaccination should only be resorted to on farms where infection in known to exist, that is, on farms where an outbreak has occurred and where depopulation was not resorted to in bringing it under control.

Infeectious Bronchitis.

This is a highly infectious respiratory disease of chickens. The disease is caused by a virus that is immunologically distinct from the virus that causes infectious laryngotracheitis.

The disorder is of widespread occurrence, frequently being noted in hatcheries and broiler plants.

Chicks between the ages of two days and three weeks are highly susceptible to this disease, though older birds may also be affected. The symptoms in infectious bronchitis are similar to those referred to under Infectious Laryngotracheitis. Post-mortem examination of semi-mature and adult birds fails to show haemorrhages in the larynx and trachea like those seen in infectious laryngotracheitis. Variable amounts of catarrhal exudate are found in the larynx and trachea. In young chicks, small pin-point haemorrhages may be noted in the larynx and trachea, and there is usually a purulent exudate present. While symptoms displayed, post-mortem findings and rapidity of spread of this disease are highly diagnostic, positive diagnosis of infectious bronchitis is dependent upon inoculation experiments.

The disease does not cause significant mortality in semi-mature and adult stock, but in young chicks, mortality from infectious bronchitis can be quite high.

Infectious bronchitis is disseminated, controlled and prevented in a manner similar to that referred to under Infectious Laryngotracheitis. Vaccination against this disease is, as yet, in the experimental stage.

Fowl Leukosis.

Fowl leukosis, until recently called fowl paralysis, is a widespread disease-complex to which both young and adult chickens are susceptible. The disease is believed to be caused by a filterable virus.

The symptoms of leukosis vary with the form in which the disease is present. In what is known as the *lymphomatosis form* (includes *neural lymphomatosis, ocular lypmhomatosis, visceral lymphomatosis* and *osteopetrotic lymphomatosis*) the principal symptoms are: paralysis of the legs (see Fig. 3), wings and neck;

depigmentation of the iris, derangement of the pupil and blindness; dropsy and in the last listed sub-form, irregular thickening of the long bones. In the forms known as *erythroblastosis, granuloblastosis* and *myelocytomatosis* the principal symptoms are: paleness or yellowish color about the head, listlessness and diarrhoea.

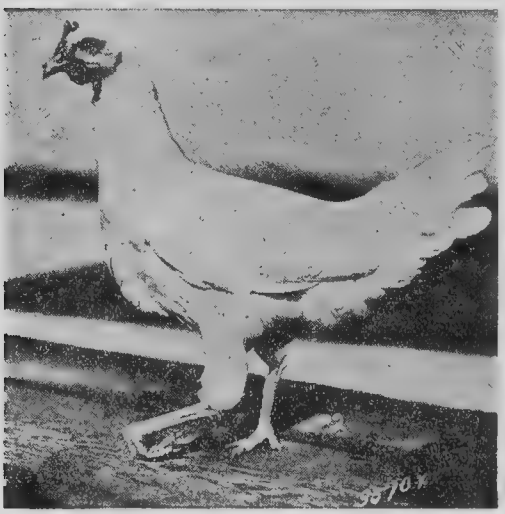

Fig. 3.—Leg paralysis due to Leukosis

Post-mortem examination of fowls afflicted with the *lymphomatosis* type of the disease reveals swollen, discolored sciatic (leg) and brachial (wing) plexuses (nerves), enlargement of the liver, spleen and kidneys, tumor-like masses involving the visceral organs (see Fig. 4) and gross alterations of the skeleton. Similar examination of birds affected with the other forms of the disease reveals watery blood, enlargement of the liver, spleen and kidneys, and tumor-like masses involving the visceral organs (see Fig. 5).

Fig. 4.—Normal (A) and abnormal (B) sciatic nerves in fowl affected with leukosis. (After California Agricultural Experiment Station.)

Fig. 5.—Liver and spleen from a fowl affected with leukosis on the left. Normal liver and spleen on the right.

Very little is known regarding the mode of spread of fowl leukosis. Transmission by contact and through the egg has been suggested. However, as yet, insufficient evidence has been produced to warrant general acceptance of this view.

Fowl leukosis constitutes a serious menace to the poultry industry today. Losses from this malady are enormous.

No treatment for fowl leukosis is known. Losses from this malady may be minimized by:

(1) Purchasing hatching eggs, chicks and adult stock only from sources that are known to be free from fowl leukosis. By purchasing new stock only from such sources, one can avoid bringing the infection on one's place.

(2) Breeding for resistance to fowl leukosis. On pedigree poultry farms, family resistance should be used as the basis of selection. On commercial poultry farms, the ability of an individual to live one or more years without showing indications of leukosis infection should constitute the basis of selection for resistance.

(3) Disposing of all birds that show symptoms of fowl leukosis as soon as noticed. By following such a procedure, spread of the malady can be checked.

BACTERIAL DISEASES

Some of the most common and economically important diseases of poultry are caused by bacteria. (Bacteria are one-celled microscopic plants.) The more important diseases in this group are: pullorum, fowl tuberculosis, fowl typhoid, fowl cholera, coryza, botulism, and omphalitis.

Pullorum.

Pullorum disease, formerly called bacillary white diarrhoea, is primarily a disease of baby chicks. It is also found in mature hens. In the chick the disease assumes an acute, highly fatal form, whereas in the adult fowl the disease is of a chronic nature.

Pullorum disease is caused by the bacterium *Salmonella pullorum.* In the hen this bacterium often becomes localized in the ovary. Many of the eggs laid by pullorum-diseasd hens are infected with the causative organism. Infected eggs, when hatched, produce infected chicks. Infected chicks pass the pullorum germ in

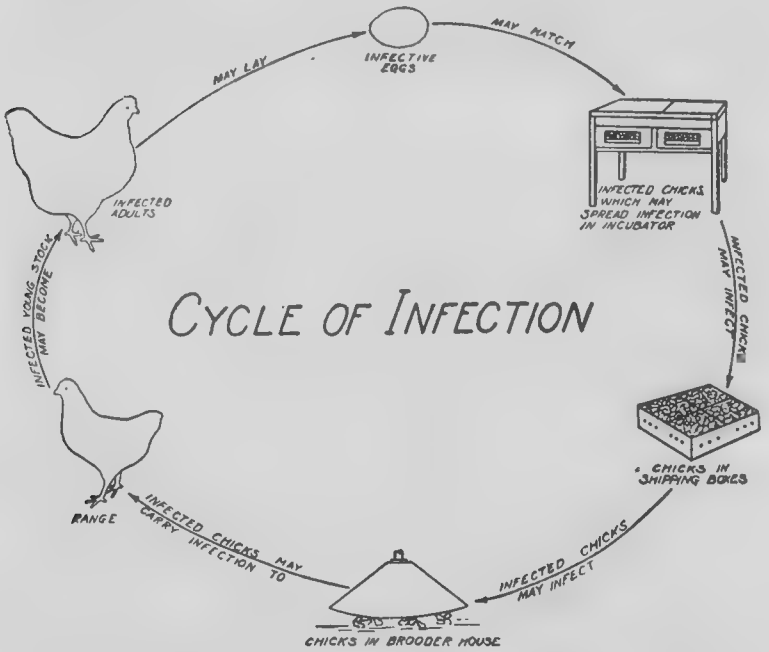

Fig. 6.—Cycle of infection of pullorum disease. (After Massachusetts Agricultural Experiment Station.)

their droppings and hence are capable of infecting other chicks, in the incubator, in shipping boxes, and in the brooder house. Many infected chicks die during the course of the disease, but some survive and grow to maturity with the infection localized in the ovary. Survivors (known as "carriers") transmit the disease through the egg to their progeny and thus the malady is perpetuated in a flock (see Fig. 6).

Pullorum disease may affect both chickens and turkeys. The disease is more or less widespread, occurring wherever poultry are kept.

Factors which predispose chicks to this disease include chilling, overheating and withholding feed.

The symptoms of pullorum disease in baby chicks are not very definite. Some chicks die so soon after hatching that no symptoms are displayed. Others present a picture of general dejection; they stand "huddled" in cornors or near the stove, with closed eyes and drooping wings and they frequently chirp plaintively, emitting shrill cries as if in pain. Diarrhoea, with "pasting up" behind, is often evident. Death usually occurs within two or three days. Heaviest losses occur during the first ten days after hatching, but mortality may continue to be quite high up to three weeks. After this period, the disease tends to change to the chronic form.

Mature fowls usually do not show any outward sign of infection.

Port-mortem examination of chicks dying soon after hatching shows no significant changes. Chicks surviving for a longer time, however, may show nodules in the heart and lungs, and the presence of a large amount of unabsorbed yolk. A definite diagnosis, however, can only be made by bacteriological examination, as similar internal appearances may be caused by chilling, overheating or improper feeding.

Fig 7A.—Normal ovary of laying hens. (After Storrs Agricultural Experiment Station.)

In adult fowls the commonest post-mortem finding is misshapen, greenish or leaden-colored yolks in the ovary (see Fig. 7). In cases where the heart is involved, pericarditis (inflammation of the heart sac) and the presence of excess pericardial fluid may be evident. The heart may be enlarged and noduled.

In chicks, mortality

from pullorum varies greatly, at times approaching 100%. In adult fowls mortality from pullorum is relatively light. Pullorum disease causes decreased egg production and decreased hatchability. Economic losses from this malady are great.

Fig. 7B.—Pullorum infected ovary of laying hen. (After Storrs Agricultural Experiment Station.)

Control of an outbreak of pullorum disease in chicks may be effected by:

(1) killing, at once, all chicks showing symptoms of the disease;

(2) preventing contamination of the feed and water by excreta;

(3) cleaning and disinfecting the brooder house and equipment at least every other day.

Now while it is true that pullorum disease in chicks may be controlled to some extent by following plans such as the one just outlined, prevention of losses from this disease involves: the identification of infected birds by a reliable pullorum test, the removal of infected birds from the breeding flock, the cleaning and disinfection of the premises, and the observation of rigid measures of sanitation and disinfection during incubation.

There are at least three reliable methods of testing adult birds for pullorum disease: (1) the tube-agglutination test, (2) the rapid-plate serum test, (3) the rapid whole-blood stained-antigen test. The tube-agglutination test and the rapid-plate serum test are conducted in the laboratory, a sample of blood being taken at the farm from each bird on the premises and shipped to the laboratory where the test is performed. The band numbers of all reacting birds are sent to the flock owner, who is then able to sort out and dispose of his "carriers." The rapid whole-blood stained-antigen test is conducted at the farm. This test has marked advantages over the two previously referred to tests in that (1) it is considerably less expensive, (2) it is better adapted to application under conditions where shipping distances to the central laboratories are great, and (3) in that it necessitates only one handling of the fowls, since it is possible to remove reacting birds as the test is being conducted. While no one of these three tests is considered 100% accurate in detecting "carriers", nevertheless, from the standpoint of reducing losses due to pullorum disease, they are of inestimable value. The consensus at the present time as to the

comparative accuracy of the tests is that they are equal when conducted by qualified technicians.

Pullorum disease, as previously indicated (see Fig. 6), is readily spread from one chick to another at hatching time. This is especially true in forced draft incubators. In order to arrest this mode of spread of infection, incubators should be fumigated.

Formaldehyde has been found very effective in incubator disinfction. Briefly the procedure is as follows: Compute the number of 100 cubic feet of air space in the incubator. Next, calculate the quantities of formalin and potassium permanganate needed on the basis of 35 cubic centimeters formalin (1⅛ oz.) and 17.5 grams (6/10 oz.) potassium permanganate for each 100 cubic feet of air space to be fumigated. Now place the measured amount of potassium permanganate in an enamel pan or pyrex dish large enough to hold about 10 times the quantity of material to be used. Set this pan in the bottom of the incubator. For large incubators more than one pan may be necessary. Next, pour the computed amount of formalin over the permanganate crystals and close the incubator door as quickly as possible. Leave the incubator door closed for three hours. At the end of this time air out the machine. Excess formaldehyde may be neutralized by sprinkling strong ammonia water on the walls of the machine.

In connection with formaldehyde fumigation it is recommended that a double strength (70 c.c. formalin, 35 grams potassium permanganate per 100 cubic feet) fumigation be given on the 20th day of incubation and that a single strength (35 c.c. formalin, 17.5 grams potassium permanganate per 100 cubic feet) fumigation be given when the chicks are 10-20% hatched. Single strength formaldehyde fumigation will not harm the chicks. It is further recommended that during the fumigation period humidity in the incubator be maintained at 70%. Operators are cautioned against fumigating machines that contain eggs which have been incubating just 24 to 72 hours. Embryos of this age are injured by double strength fumigation.

Poultrymen who desire to eradicate pullorum disease from their flocks and who wish to keep their flocks free of pullorum disease should:

(1) Eliminate "carrier" birds by blood testing (the blood testing work in Alberta is done by the Provincial Department of Agriculture). The first test should be taken shortly after the pullets come into lay. If any reactors are detected they should be disposed of immediately and the remaining birds should be retested at thirty-day intervals until such time as two consecutive negative tests are obtained. Once the flock has been shown to be free of pullorum reactors on two consecutive tests, testing at twelve-month intervals is all that is necessary. Following the removal of each lot of reactors, the premises should be thoroughly cleaned and disinfected.

(2) Purchase eggs for hatching, baby chicks and new stock

only from sources that are known to be free from pullorum infection.

(3) Maintain sanitary conditions on the farm, particularly in the incubator and in the quarters used for brooding and rearing young stock.

Fowl Tuberculosis.

Fowl tuberculosis is a chronic infectious disease that may be contracted at any age, but, because of its chronic nature, symptoms are usually not observed in birds less than one year old.

Tuberculosis in poultry is caused by the micro-organism *Mycobacterium tuberculosis avium.* The liver, spleen, and intestines are the principal parts affected by the disease, the lungs and bones are occasionally affected. The causative organism is passed in the faeces of infected birds. It is also expelled into the air by birds possessing active lung lesions.

Besides chickens and turkeys, other classes of livestock, such as swine, are susceptible to avian tuberculosis. The disease is more prevalent in the prairie provinces that in the other provinces of Canada.

As previously stated, symptoms of tuberculosis in poultry are usually only apparent in birds that are over one year old. The principal symptoms of tuberculosis in poultry are: extreme loss of weight, lameness, swollen joints, diarrhoea, sunken face, paleness about the head, ruffled feathers and drowsiness.

Post-mortem examination of birds affected with tuberculosis reveals grayish or yellow tubercles in the liver, spleen and intestines (see Fig. 2). Occasionally similar nodules are found in the bones, trachea (windpipe), lungs, ovary and mesenteries. The nodules themselves are of cheesy consistency and in the liver and spleen may vary in size from a pinhead to a small marble, while on the intestine they may be as large as an egg. The liver and spleen lesions are usually raised and are easily removed. Many of the tubercles on the intestine open into the bowel. These intestinal lesions give off countless tuberculosis bacilli into the intestine and thus the droppings become polluted with the disease-causing organism. While the above-listed lesions are indicative of tuberculosis, positive diagnosis is dependent upon bacteriological examination of affected tissues.

The most important factor in the spread of avian tuberculosis is the droppings of infected birds. In lung infections, the disease may also be spread by way of mouth. Poultry yards that are contaminated with excreta of infected birds may act as a source of infection to healthy birds for several years, as the germ is highly resistant to external influences. The bacillus is, however, readily destroyed by direct sunlight. Tuberculosis may be introduced into a flock mechanically, i.e., on the shoes of visitors, on returned ship-

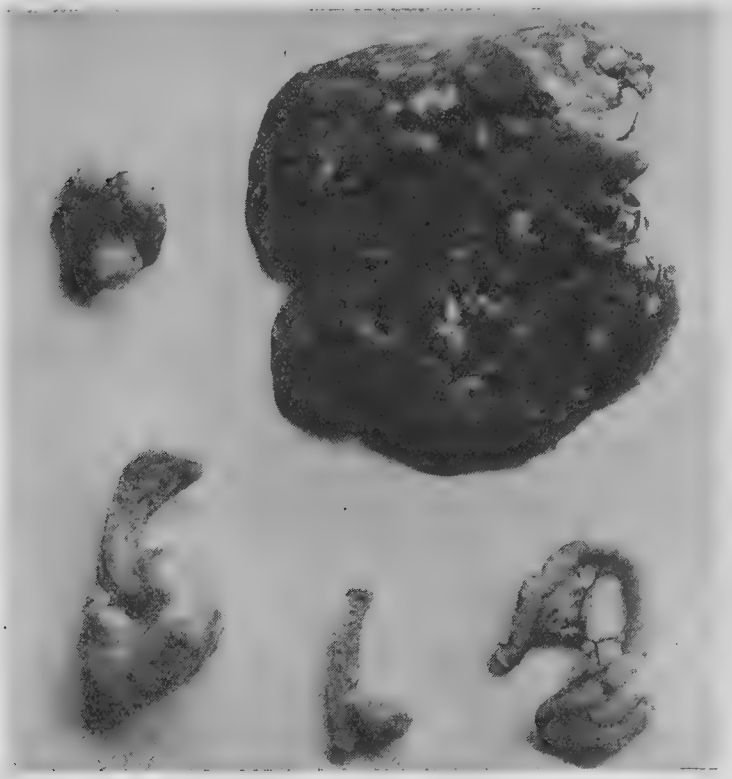

Fig. 8.—Liver, spleen and sections of intestine taken from a bird affected with fowl tuberculosis.

ping crates, on feed sacks, etc., or through the purchase of infected stock.

Mortality from this disease varies considerably, depending on the degree of infection. Losses are confined chiefly to birds more than one year old.

There is no treatment for avian tuberculosis. Once the disease is detected in a flock, rigid measures must be adopted if the disease is to be brought under control. Where possible, a tuberculin test should be made. If this test shows that the infection is high, the best procedure is to destroy and burn the affected birds, and sell the rest of the birds for meat purposes. Following depopulation, the houses and equipment should be thoroughly cleaned and disinfected and the ground over which the affected birds have ranged should be subjected to frequent cultivation, so as to expose the tuberculosis germ to the sun's rays for destruction. In connection with the disinfection of the houses and equipment, water containing 3% lye will be found inexpensive and effective. Where it is found

necessary to depopulate a poultry farm because of tuberculosis, no poultry should be housed in the same buildings, or allowed to range over the ground previously used, until at least one year has elapsed.

Where the tuberculin test has shown that the infection is light, affected birds should be disposed of and the houses and equipment should be thoroughly cleaned and disinfected. The remaining birds should not be permitted to run over the old range until it has been thoroughly cleaned up by frequent cultivation. Furthermore, in order to lessen the chances of bringing the infection from the yards into the houses, the attendant should wear rubbers in the yards and remove them before entering the houses.

Where it is not possible to have a tuberculin test made, as is the case in this Province, one is well advised to treat the flock as he would if he knew by the tuberculin test that the infection actually was high.

To avoid mechanical introduction of tuberculosis, visitors should be required to disinfect their shoes before being permitted to walk about the premises, returned shipping crates should be washed up and disinfected immediately on arriving back on the premises, feed sacks should be disinfected before being used for hauling feed about the farm, and new stock should only be purchased from flocks known to be free from tuberculosis.

Since swine are highly susceptible to avian tuberculosis, farmers are cautioned against permitting their pigs to run with poultry. The precaution is particularly applicable in Alberta where tuberculosis infection in poultry is quite prevalent.

Fowl Typhoid.

This is an infectious disease. In young stock the disease is acute in nature, whereas in adult stock both acute and chronic forms are known to occur.

Fowl typhoid is caused by the micro-organism *Shigella gallinarum*. In an infected hen this bacterium is often present in the ovary. The disease is transmitted from hen to chick through the egg in a similar manner to that referred to in the discussion of pullorum disease (see Fig. 6).

Both chickens and turkeys are susceptible to fowl typhoid and the disease is more or less widespread in occurrence. The disease is encountered most commonly in late summer.

The principal symptoms of fowl typhoid are: loss of appetite, sleepiness, droopiness, paleness about the head, yellowish or greenish diarrhoea and high fever. In the acute form the disease runs its course in from 2 to 10 days.

Post-mortem examination of birds affected with the acute type of the disease reveals a greenish bronze colored liver, watery blood, enlarged spleen and kidneys and frequently grayish spots on the liver and heart. In the chronic form, in addition to these changes,

ovaries similar to those infected with pullorum disease (see Fig. 7) and the presence of serous fluid in the pericardium (heart sac) and the body cavity are often noted. Positive diagnosis of fowl typhoid rests on bacteriological examination of affected birds.

The droppings of infected birds contain large numbers of the typhoid organism and hence serve as a source of infection for healthy birds. The typhoid bacterium is not as resistant to external influences as the germ causing fowl tuberculosis.

Mortality from this disease varies grately, in some instances very few losses are sustained, while in other instances entire flocks are wiped out.

Control of an outbreak of typhoid in chicks may be effected by:

(1) killing, at once, all chicks showing symptoms of the disease;
(2) preventing contamination of the feed and water by excreta;
(3) cleaning and disinfecting the brooder house and equipment at least every other day.

There is no cure for fowl typhoid. In flocks where it has been detected, steps should be taken immediately to eradicate the malady. A pullorum test (which also detects birds harbouring the typhoid organism) should be conducted at monthly intervals until two consecutive tests show no reactors. All reactors should be disposed of as soon as detected. Following each test in which reactors are found, the houses and equipment should be thoroughly cleaned and disinfected. Under no cirmumstances should the non-reacting birds be permitted to run over their old range, for this would only result in further infection. The old range should be cleaned up by cultivating frequently, so as to expose the typhoid germ to the elements for destruction. One season's cultivation should be sufficient.

Fowl Cholera.

Fowl cholera is an acute infectious disease. Occasionally it occurs as a chronic disease, though it is generally characterized by sudden death and high mortality.

The disease is caused by the micro-organism *Pasteurella avicida*. In infected birds this organism is found in large numbers in the blood and in various organs of the body. It produces a septicemia, i.e., a blood poisoning.

Fowl cholera is widespread in occurrence and is known to affect both chickens and turkeys. The disease is encountered most frequently during the winter months.

The symptoms of this disease vary widely. The onset is usually sudden. Birds are seen walking around the pen and in a short time are found dead. Often the first indication of the presence of this disease is that birds are found dead in the nests or under the roosts. As the outbreak progresses the disease assumes a less acute form. In the less acute form, the symptoms commonly noted are: loss of

appetite, droopiness, sleepiness, darkness about head, yellowish or greenish diarrhoea, restrained breathing, high fever, increased thirst and oedema of the wattles.

Post-mortem examination of birds that die suddenly from fowl cholera often shows no changes present. However, similar examination of birds that have been affected for several days with this disease reveals: accumulation of sticky mucus in the mouth, congestion of visceral organs, pin-head sized necrotic areas in the liver, friable liver, haemorrhages of various sizes on the heart and mesenteries, straw-colored fluid in the pericardium (heart sac), inflammation and pin-point haemorrhages in the upper portion of the intestines and cheesy yellow deposits among the intestines. In the chronic form, cholera has been known to cause swollen joints, bumblefoot, peritonitis (inflammation of membrane covering viscera) and ascites (accumulation of serous fluid in the abdominal cavity). Positive diagnosis of fowl cholera is dependent upon bacteriological examination of the blood of infected birds.

As with fowl tuberculosis and fowl typhoid, the droppings from infected birds constitute the most important factor in the spread of this disease. The germ causing the disease gains entrance to the bird's body by way of the respiratory and digestive tracts.

In the acute form this disease may practically wipe out a flock in the course of a week or two. Losses from chronic cholera are light.

Treatment of affected birds is useless. However, once the disease is detected, vigorous measures of control should be adopted. Dead and ailing birds should be disposed of immediately by burning. All houses and equipment should be thoroughly cleaned and disinfected. Drinking utensils should be disinfected daily. Feed troughs should be protected from faecal contamination. The old range should not be used again for poultry until it has been cleaned up by cultivating for at least one season.

Coryza.

The terms, cold, roup, catarrh, influenza and coryza have been applied by poultrymen more or less interchangeably in referring to any inflammation of the mucous membranes of the upper respiratory tract. Coryza, however, is the proper designation for an infectious cold. The consensus at the present time as to the cause of coryza is that one or more specific bacterial agents are capable of producing the condition, and that several other agents play important roles in the production of the symptoms.

The first birds to contract coryza are those of low vitality. Hence any factors that tend to affect vitality adversely must be considered as predisposing to coryza. Overcrowding, improper ventilation, improper feeding, lack of vitamin A, infestation with lice, mites, worms or coccidia, and the undue exposure to inclement

weather, drafts, dampness or insanitary conditions, all fall in this category and should, therefore, be avoided.

Early stages of coryza are characterized by watery eyes and a serous discharge from the nostrils. As the disease progresses, the nasal discharge becomes thick, sticky and foul smelling (probably due to secondary infection), and tends to form yellowish crusts about the nasal openings. With the extension of the inflammation, the sinuses become filled with mucus, which, being unable to drain away, changes to a cheesy material. This cheesy material accumulates, causing the sinuses to bulge (see Fig. 9). Similar cheesy material also accumulates in the cleft in the roof of the mouth. In very advanced cases, cheesy patches are often found in the mouth itself. These cheesy patches in the mouth are readily distinguished from those noted in the diphtheritic form of fowl pox in that they are not so adherent as are those caused by fowl pox. In cases where the trachea (windpipe) is involved, difficulty in breathing is experienced. This is due to an accumulation of mucus in the trachea. Severely affected birds usually sit quietly, with feathers ruffled, show little or no inclination to eat, wheeze and sneeze at regular intervals and frequently shake their heads in an effort to dislodge mucus.

Fig. 9.—Watery eye and swollen sinus in a fowl affected with coryza. (After Iowa Agricultural Experiment Station.)

Post-mortem examination shows no characteristic lesion other than those described above.

Coryza is one of the most common ailments affecting poultry. While mortality losses from this disorder are of a relatively low order, losses due to poor growth in growing birds and poor egg production in laying birds are very great.

Preventive measures involve giving due consideration to all factors that may have deleterious effects on vitality and so predispose to coryza. Overcrowding should be avoided. Lightweight layers should have three square feet of floor space, heavyweights four. Poultry houses should be properly ventilated. Openings should be so arranged as to permit of a steady circulation of air through the house, the moist, foul air being drawn off and replaced

by fresh air. Drafts should be avoided, especially is this true of drafts over the roosts. Birds should not be unduly exposed to inclement weather, and should be kept free of lice, mites, worms and coccidia, for all tend to lower vitality and predispose to disease. Further, care should be taken to see that the ration fed is well-balanced and that it is properly administered.

Even under the best of management, colds may develop. Accordingly one should watch his birds very closely. On the slightest sign of infection, the predisposing causes should be determined and corrected. Next, all affected birds should be removed from the main flock. Following their removal, daily disinfection of water utensils and nightly spraying of birds on the roosts with emulsified eucalyptus oil, or a mixture of lysol, kerosene and camphor in proportions of 1:1:2, should be practised. This will tend to curtail further spread of infection in the main flock.

As far as success in treating affected birds is concerned, much depends on the stage at which the disease has reached. Mild cases are readily cleared up, while very advanced cases are seldom worth bothering with, as recovery is doubtful, even with the best of care. Birds that have been removed from the main flock because of infection should be placed in a warm, dry, properly ventilated building, and should be given a well-balanced ration. If the birds are known to be infested with lice, mites or worms, they should be treated for these (see External and Internal Parasites). Then in order to clear up any sluggishness of bowel movement that might be present, Epsom salts, at the rate of three-quarters of a pound per hundred birds should be administered. Next, the birds should be sprayed at frequent intervals with a good inhalent (see above). Spraying should be continued until such time as the trouble has cleared up. Water utensils should be disinfected daily.

Besides following this general plan, the eyes, nostrils, sinuses (after being incised), cleft of the mouth and mouth proper of birds in the sick-house should be thoroughly washed out with a 10% solution of baking soda, a 4% solution of boracic acid, or a 2% solution of potassium permanganate so as to permit free breathing. Drops of 4% nitrate or 10 to 15% argyrol or 5% iodine should next be placed in the eyes, nostrils, sinuses and on mouth cankers. This individual treatment should be repeated daily until no more pus accumulates. Birds that are badly affected, and those that do not improve readily, should be destroyed.

An individual treatment that is being used extensively, especially by turkey raisers, consists of draining the sinuses by means of a 10 to 20 cubic centimeter syringe with a 16 gauge, 1½″ needle, and introducing into the drained sinuses by means of another syringe 1 c.c. of 4% silver nitrate or 1 c.c. of 10 to 15% argyrol. This treatment is only effective when the material in the sinuses is liquid or semi-liquid. If the material in the sinuses is of cheesy consistency incision is necessary.

A further individual treatment that is recommended highly consists of correcting the predisposing cause, isolating affected birds, removing mouth obstructions, and twice submerging the heads of affected birds in sodium hypochlorite solution for about one minute (until inhalation is evidenced by bubbling from the mouth or nostrils). Sodium hypochlorite solution is made by mixing one part of sodium hypochlorite (1% avoilable chlorine) with two parts of water. Severely affected birds are best destroyed.

Botulism.

Botulism, also known as limberneck, is an acute or sub-acute intoxication caused by exotoxins (poisonous substances secreted or excreted by bacteria), produced by the microscopic *Clostridium botulinum,* types A and C. The organisms themselves do not affect the fowl adversely, but the exotoxins produced by them during the process of their multiplication in decaying matter do. Minute amounts of the toxins (types A and C) are sufficient to produce severe toxic effects and only slightly more are lethal.

Outbreaks of botulism occur chiefly during warm weather, as it is during such weather that putrefaction is going on most rapidly. Both chickens and turkeys are susceptible to botulism.

The principal signs of botulism are: sleepiness, limpness of neck and wings (see Fig. 10), incoordination, prostration, and looseness of the feathers. Post-mortem examination reveals no changes in the organs. The symptoms displayed by affected fowls are the chief means of diagnosing botulism.

Fig. 10 — Fowls affected with botulism. (After Western Washington Agricultural Experiment Station.)

Treatment of badly affected birds is of little avail. However, Epsom salts given at the rate of two pounds per hundred birds will aid in flushing the toxins out of the intestinal tract of less severely affected birds, and as a consequence effect their recovery. Following the purge, the houses should be cleaned so as to remove toxin eliminated into the litter. To prevent further spread or recurrence of the trouble, the source of the toxins must be found and removed. In this connection, the premises should be carefully searched for decomposing organic material, which as previously stated, constitutes the source from which birds obtain the toxins.

Omphalitis.

Omphalitis, or navel ill, is a disease of chicks and poults in which the navel appears abnormal. It is the result of bacterial infection of the navel at hatching time. A number of different micro-organisms are capable of causing the condition. The abnormality is ofttimes encountered in commercially hatched chicks and poults.

The principal symptoms of the disorder are: pus about the navel, puffiness in the abdomen, and the presence of diarrhoea. Internal examination reveals: putrefaction about the navel, unabsorbed yolk, yellowish liver, and distention of the gall bladder.

The disease shows up the second or third day after hatching. Heaviest losses occur between the third and sixth day after hatching. There is no effective treatment for this disorder. It may, however, be prevented by observing proper incubator sanitation. This entails thoroughly cleaning and disinfecting the incubator before the hatching season commences and fumigating the incubator with formaldehyde gas before and during each hatch. For details on incubator fumigation see Pullorum disease.

PROTOZOAN DISEASES

Protozoan diseases are diseases caused by minute organisms called protozoa. (Protozoa are one-celled organisms, the smallest in the animal kingdom.) The more important diseases in this group are coccidiosis and blackhead.

Coccidiosis.

Coccidiosis is an intestinal disease caused by microscopic parasites known as coccidia. In young birds the disease is usually acute in nature, while in old birds the disease generally follows a chronic course.

Seven species of coccidia are capable of infecting the domestic fowl. They are: *Eimeria tenella, E. necatrix, E. mitis, E. acervulina, E. praecox, E. maxima* and *E. hagani.* Generally speaking, only two of these species are decidedly pathogenic: *E. tenella* affects the caeca (blind pouches of the intestine) of chicks, causing acute coccidiosis; *E. necatrix* affects the duodenum (U-shaped part of the small intestine) and mid-portion of small intestine of fowls of four months of age and over, causing, in some instances, acute coccidiosis, but more often chronic coccidiosis.

Large numbers of coccidia are passed in the droppings of infected birds. The organisms are voided in the stage of their life cycle known as "oocysts." Oocysts are not directly infective to birds, but, under favourable conditions of warmth and moisture, they undergo a change (sporulation) in twenty-four to seventy-two hours, giving rise to what are known as "sporulated oocysts." Sporulated oocysts are highly infective to susceptible birds. Upon

being ingested by a susceptible fowl, they (sporulated oocysts) give rise in the intestine to small spindle-shaped forms called "sporozoites." The sporozoites penetrate the cells lining the intestine or caeca (depending on the species) and give rise to forms known as "schizonts." The schizonts undergo a series of nuclear divisions (schizogony) and eventually split up into a number of nucleated fragments or "merozoites." These merozoites, upon release, invade other cells to give rise to more schizonts, which, in turn, undergo schizogony to give rise to more merozoites. This schizont to merozoite to schizont development is repeated several times (the number of times varies with the species). Then a change occurs. Instead of more schizonts being formed from merozoites, male and female forms are produced. These, upon release into the intestinal tract, unite to form oocysts, which, as stated earlier, are voided in the droppings and are not infective until sporulated by the action of heat and moisture. Under average conditions, the species of coccidia infecting the domestic fowl take from four to ten days to complete their life cycle.

It is of interest to note here that each intestinal cell invaded by a sporozoite, or by a merozoite, is destroyed during the course of the development that ensues. Where extensive destruction of cells has occurred the absorptive area of the intestine is greatly reduced, and as a result absorption of nutrients is interfered with. Furthermore where a large number of cells in one area are destroyed, haemorrhage of blood vells in that area takes place with the loss of much blood into the intestine.

Coccidiosis is of widespread occurrence. The incidence of this disease is greatest during the spring and summer months. This, of course, is due to the fact that during those months, conditions are ideal for the sporulation of dormant oocysts. Chickens of three to twelve weeks of age are highly susceptible to coccidiosis. In chickens of this age, the disease is acute in form, and causes heavy losses. Older chickens may also be affected with coccidiosis. However, in older chickens, the disease is usually chronic in form, though acute outbreaks are not uncommon.

The principal symptoms noted in outbreaks of acute coccidiosis are: loss of appetite, emaciation, weakness, droopiness and passage of blood in the faeces. Chronic coccidiosis is manifested by: loss of appetite, progressive emaciation, sluggishness, paleness and leg weakness.

Post-mortem examination of birds afflicted with acute coccidiosis shows enlargement of caeca, thickening and inflammation of the walls of the caeca and the presence of blood in the caeca (see Fig. 11). In cases where the disease has been less acute, cheesy blood-stained masses are often found in the caeca. Where the disease has run a chronic course, changes are usually confined to the duodenum. The intestinal walls are thickened and inflamed and small haemorrhages are often noted. Positive diagnosis of

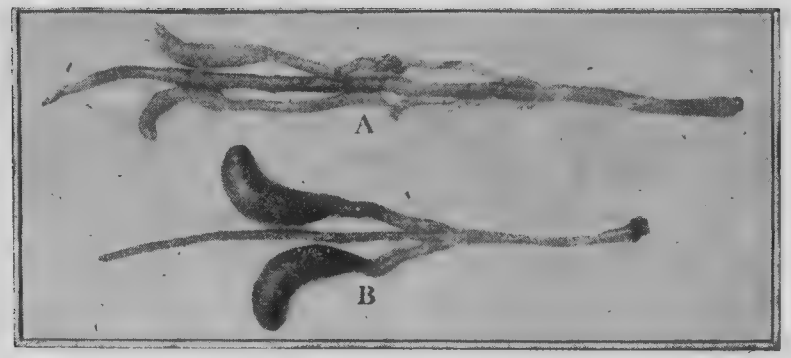

Fig. 11.—(A) Normal caeca. (B) Caeca from bird affected with coccidiosis. (After Idaho Agricultural Experiment Station.)

coccidiosis is dependent upon the finding of coccidia in intestinal scrapings. This, of course, necessitates the use of a microscope.

The droppings of infected birds are the chief source of infection to other birds. Ground, houses and equipment contaminated with faeces from infected birds may serve as a source of infection to susceptible birds for as long as two years. Birds that have recovered from the disease, and birds afflicted with the disease in chronic form, are a constant menace to the well-being of other birds in the flock, for they are continually passing oocysts in their droppings. The disease may be introduced into a flock through the purchase of infected birds or by permitting birds in the flock to have access to contaminated ground, houses or equipment.

Mortality from coccidiosis is often quite high, especially where control measures are not instituted promptly. Losses in young chicks usually come all at one time, whereas losses in adult fowls are generally spread over a longer period.

An outbreak of acute coccidiosis in chicks or adult fowls may be effectively brought under control by carrying out the following program. Confine all affected birds to a clean house for a period of ten days. Change the litter daily during the confinement period. This is necessary in order to prevent any of the oocysts passed by the birds reaching the infective stage. See that the feed and water are not contaminated with faecal material. Keep the birds away from the damp, dropping-polluted litter about the water fountain by placing the fountain up on a wire stand. If reinfection has not occurred by the tenth day, all the coccidia in the birds will have been voided as oocysts and the disease will have been brought under control.

During the first three days of confinement, the regular mash should be replaced by mash of the following formula: corn meal 30, barley 20, bran 10, buttermilk powder 40. This high buttermilk content mash will tend to flush out the birds. Following the third

day, the feeding of the regular mash can be continued. Grain should be fed sparingly while the birds are on this flushing mash.

A further treatment for acute coccidiosis and one which is recommended widely is what is known as the molasses bran treatment. Feeding and management details for this treatment are as follows:

1. For three consecutive days give as the sole forenoon feed a mixture of molasses and bran (15% molasses, 85% bran). In the afternoons feed the usual mash and scratch grain.

2. During the three day treatment period and for four days immediately following—a full week in all—keep the birds confined to the brooder house and clean out the brooder house thoroughly each day.

After the confinement period has elapsed, the birds should be let out on clean range, i.e., on range to which poultry have not had access during the previous two years. Under no circumstances should the birds be permitted to run over their old range, for this would only result in reinfection.

The contaminated range may be cleaned up by cultivating. No poultry should be allowed on the old range until at least two years time has elapsed.

Chronic coccidiosis is best controlled by frequent culling of sickly birds, and by the observation of proper sanitation on the farm.

Losses from coccidiosis may be prevented by:

(1) Rearing young stock on clean range away from old stock.

(2) Observing proper sanitation on the farm, i.e., cleaning the dropping boards, houses and yards regularly; avoiding dampness about the water fountains; avoiding puddles on the range, etc.

(3) Culling out sickly birds as soon as noticed.

(4) Keeping the flock in good vigour by proper feeding and management. This is important because birds that are down in vigour are known to be more susceptible to coccidiosis than those that are vigorous and healthy.

At the present time experimental work is being done on the value of sulfa compounds in preventing coccidiosis. The results obtained to date are encouraging; however, practical recommendations regarding the use of these drugs in the prevention of coccidiosis cannot, as yet, be made.

Blackhead.

Blackhead, also known as infectious enterohepatitis, is an acute or chronic infectious disease caused by the protozoan organism *Histomonas meleagridis*. The disease is of widespread occurrence, affecting principally turkeys, though chickens may also be affected. In turkeys of one to three months of age, the disease is rapid in its course and highly fatal. In adults the disease follows a chronic course, losses being but occasional.

The main symptoms of blackhead in young turkeys are: loss of appetite, increased thirst, poor growth, emaciation, weakness, drowsiness, ruffled feathers and drooped wings. Orange-brown diarrhoea may also be evident. The principal symptoms of this disease in adult birds are: progressive emaciation, sunken eyes, sluggishness and diarrhoea. Chickens affected with blackhead usually do not show any outward evidence of infection. Birds that recover from blackhead remain "carriers" of this disease.

Autopsy findings in blackhead are confined to the liver and caeca (blind pouches of the intestine). The livers of affected birds are often enlarged and have round, sunken, ulcer-like areas of pink or yellowish color over their surfaces (see Fig. 12). The caeca are usually very much enlarged and their walls are inflamed, thickened, and ulcerated (see Fig. 12). The presence of cheesy material in the caeca is also characteristic of this disease. While the symptoms and post-mortem findings described are definitely suggestive of blackhead infection, positive diagnosis is dependent upon microscopic examination of the liver and caeca of affected birds.

Fig. 12.—Liver and caeca from a turkey affected with blackhead.

Six days after infection has taken place, *Histomonas meleagridis* the causative organism, is passed in the droppings of affected birds, either in free state or enclosed in the egg of the caecal worm, *Heterakis gallinae*. In the free state the organism seldom survives more than twenty-four hours. However, when enclosed in the caecal worm egg, the organism is able to survive for a much longer period of time. The principal agents responsible for the spread of this disease are infected or carrier chickens and turkeys.

Heaviest losses from blackhead are sustained in young turkeys. Losses in adult turkeys are but occasional.

No specific treatment for blackhead is known. Where an outbreak of this disease has occurred, the following control measures are recommended:

(1) Dispose of all sickly birds.

(2) Move all apparently healthy birds to clean range or into houses with wire sun-porches.

(3) Clean and disinfect the houses and equipment regularly.

(4) Watch for further cases and dispose of them as soon as detected.

The occurrence of blackhead may be prevented by rearing young turkeys on wire for the first ten to twelve weeks and then growing them out on clean range, away from old turkeys and by keeping both young and old turkeys away from chickens or ground to which chickens have had access. Furthermore, since the blackhead organism enclosed in the egg of the caecal worm will remain alive for a much longer time than when not so protected, it should be obvious that preventive measures for blackhead should also include rigid control over caecal worm infestation on the farm (see Internal Parasites).

FUNGOUS DISEASES

There are at least three diseases to which poultry are susceptible that are caused by fungi. They are: aspergillosis, favus and thrush.

Aspergillosis.

Aspergillosis, also known as brooder pneumonia, is a respiratory disease to which both young and adult birds are susceptible. The disorder is caused by the fungus *Aspergillus fumigatus,* which is invariably present in mouldy feed and litter.

External symptoms of this disorder are: sluggishness, droopiness, loss of appetite, emaciation, sneezing, wheezing, and gasping for breath. In young birds the disease is very rapid in its course and usually fatal. In adult birds the disease is much slower to develop and less fatal. Post-mortem examination of afflicted birds reveals grayish, greenish or yellowish lesions in the trachea (windpipe), bronchi, lungs, and air sacs. Positive diagnosis of this disease is dependent upon microscopic examination of affected tissue.

In the event of an outbreak of aspergillosis the feed and litter should be checked for the presence of mould. If the feed is at fault the troughs and hoppers should be cleaned and disinfected and refilled with fresh feed, free of mould. If, on the other hand, mouldy litter is the cause of the trouble the house should be thoroughly cleaned and disinfected and replenished with mould-free litter. .5% copper sulphate solution will be found an effective disinfectant for mould.

Favus.

Favus, or white comb, is a skin disease of young and adult birds, caused by the fungus *Achorion gallinae.* The disease is transmitted from one bird to another by direct contact.

Favus is characterized by the presence of dirty-white or yellowish scaly lesions on the comb, earlobes, face and wattles. In advanced cases the head and neck may be bare. Positive diagnosis

of favus is dependent upon microscopic examination of affected tissue.

Birds afflicted with favus may be treated with formalized vaseline. Formalized vaseline is prepared as follows: melt a small amount of vaseline in a jar provided with a tight fitting cork, to the melted vaseline add 5% by weight of commercial formaldehyde, cork the jar tightly and shake until the vaseline has hardened. One or two applications of formalized vaseline are usually sufficient.

Another treatment that has been found effective consists of applying to the affected parts a mixture of one part tincture of iodine to six parts of glycerine. Two or three applications are usually necessary.

Favus is transmissible to man through skin abrasions. However, where a reasonable amount of care is exercised in handling affected birds there is little danger of contracting the disease.

Thrush.

The term, thrush, is used to designate a fungous disease of fowls in which the mucous membranes of the digestive tract are involved. Several different species of fungi are capable of causing thrush.

One species, *Oidium pullorum*, attacks the mucous membranes of the mouth, pharynx, and upper larynx, causing cankerous growths. These cankerous growths differ from those encountered in diphtheritic fowl pox in that they can be removed without causing bleeding, whereas removal of pox cankers usually causes bleeding. In cases where the mouth and pharynx are involved, eating is made difficult, and as a result affected birds lose weight rapidly. On the other hand, where the upper larynx is involved, death by asphyxiation often results. Treatment of this form of thrush is readily accomplished by removing all cankerous growths and painting the areas from which the cankers weere removed with tincture of iodine.

Two other species of fungi, *Monilia albicans* and *Monilia krusei*, attack the gullet, crop, proventriculus, gizzard, intestine, and anus, causing severe ulceration. Chicks and poults affected with this from of thrush die rapidly. Adults, on the other hand, are much slower to die. Treatment of advanced cases of this form of thrush is useless. Treatment of less advanced cases may be accomplished by giving copper sulphate (bluestone) in the drinking water. The proper dilution of copper sulphate for this purpose is 1:2000. Dilutions stronger than 1:500 should not be given as they are toxic. The 1:2000 copper sulphate solution should be kept before the birds as their only drink until the trouble has cleared up. The solution should be supplied in wooden troughs or earthenware, not in metal ware, as copper sulphate corrodes metal. During the treatment the houses and equipment should be kept scrupulously clean.

A convenient method of making up approximately a 1:2000 dilution of copper sulphate solution is as follows: Make up a stock

solution by dissolving one pound of copper sulphate in one gallon of soft water (rain water). If soft water is not available, add one teaspoon of concentrated hydrochloric acid or one cup of vinegar to the water before adding the copper sulphate. Bring the copper sulphate into solution by heating. Store solution in glass or crockery ware. To make a 1:2000 dilution add one tablespoon of the stock solution to one gallon of soft water. If hard water is used enough hydrochloric acid or vinegar should be added to same to prevent precipitation of the copper. The amount of acid required will vary with the hardness of the water. Not more than one teaspoon of hydrochloric acid should be added to each gallon of water.

Positive diagnosis of either of these two forms of thrush can only be made by microscopic examination of affected tissue.

The fungi responsible for both forms of thrush are passed in the droppings of affected birds. Susceptible birds coming in direct contact with infected droppings contract the disease.

Trouble from thrush may be avoided by seeing that feed and litter used for poultry are free of mould and by keeping houses and equipment in sanitary condition.

NUTRITIONAL DISORDERS

Nutritional disorders arise as a result of a deficiency or an excess of one or more nutrients in the diet. Among the nutritional disorders that are commonly encountered in poultry are: nutritional roup, curled-toe paralysis, rickets, perosis, gout and obesity.

Nutritional Roup.

Unlike ordinary roup (coryza), nutritional roup is not infectious but is due to lack of a specific vitamin (vitamin A) in the diet.

Nutritional roup is most often observed under conditions where rations inadequate in vitamin A are fed to birds kept in confinement or to birds that do not have access to good green range. The disorder is seldom encountered in birds on good range. This, of course, is due to the fact that good range is an excellent source of vitamin A.

The symptoms of vitamin A deficiency in young chicks are: loss of appetite, poor growth, staggering gait, soreness of the eyes, diarrhoea, and drowsiness. The manifestations of nutritional roup in young turkeys are the same as those listed for chicks except that young turkeys often show one additional symptom, namely, swelling of the sinuses. Deficiency of vitamin A in the diet of laying birds causes low hatchability of eggs, decreased egg production and eventually, cessation of egg production. The symptoms of prolonged vitamin A deficiency in adult birds are: loss of appetite, incoordinated gait, soreness and wateryness about the eyes and

accumulation of cheesy material in the eyes, swollen sinuses, impaired vision, droopiness, and sleepiness.

Post-mortem examination of afflicted chicks reveals white deposits (urates) in the kidneys and cloaca (chamber just inside vent) and similar deposits on the heart, liver and other visceral organs. Autopsy findings in affected poults are not constant. An internal examination of adult birds suffering from nutritional roup shows urate deposits in the kidneys and cloaca, and similar deposits on the heart, liver and other visceral organs and the presence of

Fig. 13.—White pustules in mouth, pharynx, and oesophagus of bird affected with nutritional roup. (After California Experiment Station.)

white pustules in the mouth, pharynx and oesophagus (gullet) (see Fig. 13). While diagnosis of advanced stages of nutritional roup can readily be made on the aforementioned symptoms and

post-mortem findings, diagnosis of less advanced stages is impossible on that basis. To diagnose less advanced stages properly one must have knowledge of the composition of the ration that is being fed and of the range conditions under which the birds are kept.

Losses from nutritional roup in Alberta are relatively serious. The loss due to death from advanced nutritional roup is relatively insignificant when compared to the loss sustained through poor growth and development of young stock, poor egg production of laying stock, poor hatchability of eggs from breeding stock and low resistance to disease in all classes of poultry.

Trouble from nutritional roup may be avoided by including an adequate amount of Vitamin A in the diet. The vitamin A requirements per hundred grams of feed (the vitamin requirements per hundred grams of feed are given, rather than per hundred pounds of feed as most vitamin tables are based on metric weight) of various classes of chickens are as follows: growing chickens—320 I.U. (International Units), laying chickens 700 I.U., and breeding chickens—1,040 I.U. The exact vitamin A requirements of corresponding classes of turkeys are, as yet, unknown. Tentative figures for turkeys are: growing turkeys—800 I.U., and breeding turkeys—1,040 I.U. per hundred grams of feed. To meet these requirements for vitamin A, the average chicken growing mash or laying mash should contain 5% alfalfa leaf meal, while the average chicken breeding mash, turkey growing mash or turkey breeding mash should contain 7½% alfalfa leaf meal. These levels, when fed in conjunction with appropriate amounts of fish oil (see Rickets) will suffice.

In addition to obtaining vitamin A from alfalfa leaf meal and fish oil, poultry obtain vitamin A from green feed, yellow carrots, and yellow corn. When an abundance of these vitamin A carrying feeds are available for feeding, the alfalfa leaf meal in the mash may be reduced materially.

Treatment of nutritional roup may be accomplished by supplying an abundance of vitamin A in the diet of the affected birds. The vitamin A content of the mash should be well in excess of the requirements, and in addition the affected birds should be given all the fresh green feed that they can consume.

Curled-toe Paralysis.

Curled-toe paralysis is a disorder encountered in chicks receiving inadequate amounts of riboflavin in their diet. The condition is characterized by paralysis of the legs and inward curling of the toes (see Fig. 14).

Besides being necessary for the prevention of curled toe paralysis, riboflavin is essential for the promotion of normal growth, egg production and hatchability.

Feedstuffs which contain substantial amounts of this vitamin include liver meal, buttermilk powder, dried whey, skimmilk powder, and alfalfa leaf meal.

The riboflavin requirements per hundred grams of feed of various classes of chickens are as follows: growing chickens — 360 μ.g., laying chickens—180 μ.g., and breeding chickens —300 μ.g. The exact riboflavin requirements of corresponding classes of turkeys are, as yet, unknown. Tentative figures for turkeys are: growing turkeys —360 μ.g., and breeding turkeys—360 μ.g.

Fig. 14.—The chick on the left received an adequate amount of riboflavin in its diet while the one on the right did not. The chick on the right is showing symptoms of curled-toe paralysis. Both chicks were four weeks old when photographed.

These riboflavin requirements may be met by including in the average growing mash 5% milk powder or 2 grams synthetic riboflavin per ton, in the average laying mash 2½% milk powder or 1 gram synthetic riboflavin per ton, in the average breeding mash 7½% to 10% milk powder or 3 to 4 grams of synthetic riboflavin per ton.

Chicks afflicted with curled-toe paralysis may be brought back to normal by placing them on a diet adquate in riboflavin and by greatly increasing their riboflavin intake for a period of about a week. The latter may be accomplished by giving the chicks three or four feeds of chopped raw liver per day or by adding a little extra synthetic riboflavin to the mash.

Rickets.

Rickets is a disorder characterized by faulty mineralization. It may be caused by (1) vitamin D deficiency, (2) calcium deficiency, (3) phosphorus deficiency, or by (4) improper balance between calcium and phosphorus in the ration.

The principal symptoms of rickets in growing birds are: staggering gait, reluctance to stand or move, enlarged joints, bent legs, soft beaks, crooked breastbones, poor growth and general unthrift. Post-mortem examination reveals beaded rib condition and piling up of cartilage at the ends of the long bones. Bone ash analysis

shows low mineral content of bones. In layers, rickets results in impaired hatchability and egg production, and in the production of thin-shelled abnormally small eggs. Rarification of bones also takes place with the result that breastbones as well as other bones become crooked.

Rickets may be avoided by providing a ration adequate with respect to vitamin D content and calcium and phosphorus content and balance. The vitamin D requirements per hundred grams of feed of various classes of chickens are as follows: growing chickens —40 I.U. (International Units), laying chickens—80 I.U., and breeding chickens—120 I.U. The calcium and phosphorus requirements of growing chicks are 1.4% calcium, 0.8% phosphorus; of laying and breeding chickens are 2.4% calcium, 1.0% phosphorus. The exact vitamin D, calcium and phosphorus requirements of turkeys are, as yet, unknown. Tentative figures for turkeys are: growing turkeys—200 I.U. vitamin D per hundred grams of feed, 1.4% calcium, 0.8% phosphorus; breeding turkeys—120 I.U. vitamin D per hundred grams of feed, 2.4% calcium, 1.0% phosphorus.

The vitamin D requirements (given above) may be met by either permitting birds to have access to direct sunlight (the ultraviolet rays of the sun act on certain substances present in the feathers and skin to produce vitamin D) or by including fish oil (containing 400 I.U. vitamin D per gram) in the mash at the proper level (1/10 to 1% in growing mashes, 1/5 to 3/5% in laying and breeding mashes). The calcium and phosphorus requirements of growing stock may be met by including 1% limestone in the average starter, and 1% limestone and 1% bone-meal in the average growing mash, plus oyster shell and bone-meal *ad lib*, after the feeding of scratch grain has been commenced. The calcium and phosphorus requirements of laying and breeding stock may be satisfied by including 2% limestone and 2% bone-meal in the average laying and breeding mash and by feeding oyster shell *ad lib*. When protein supplements of vegetable origin are used in compounding poultry mashes, 1% bone-meal should be included in the mash for each 5% to 10% vegetable protein used. This is necessary in order to compensate for the low mineral content of protein supplements of vegetable origin.

Where trouble from rickets is encountered, the mineral balance of the ration should be checked, and if necessary, corrected. Then the vitamin D content of the mash should be increased to approximately twice the level required. Following correction of the disorder, the vitamin D content of the mash should be reduced to the proper level.

Perosis.

Perosis, also known as slipped tendon, is a disorder that is frequently noted in growing birds, particularly in growing birds raised in confinement. It is prone to occur on rations that contain

excessive amounts of calcium or phosphorus or inadequate amounts of manganese, choline or biotin.

The condition is characterized by bowing or twisting of the legs, slipping of the Achilles' tendon (large tendon of the hock joint) from its normal position and enlargement and flattening of the hock joint (see Fig. 15).

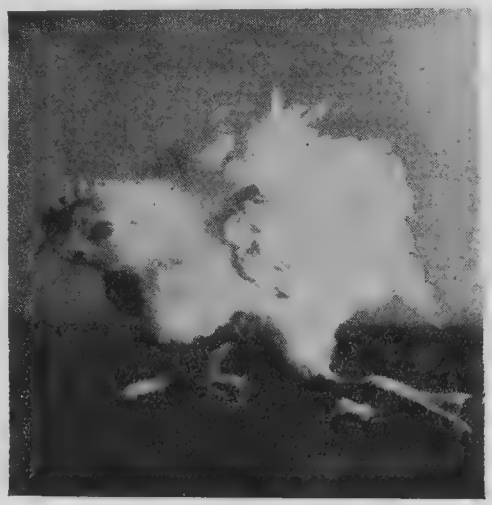

Fig. 15.—Chick affected with perosis.

There is no method of treating birds affected with perosis. Control of losses from this disorder must therefore be effected through prevention. Perosis may be avoided by including proper amounts of calcium (1.4%), phosphorus (0.8%), manganese (0.005%), choline (chickens .15%, turkeys .25%) and biotin in the ration.

How to obtain these levels of calcium and phosphorus is explained under Rickets, paragraph 4. To meet the manganese requirement (given above), ¼ to ½ of a pound of manganese sulphate should be added to each ton of ration. The choline and biotin requirements may usually be met by including generous amounts of liver meal (2—4%), shorts or middlings (10-20%), bran (5-10%) and oats (10-20%) in the ration. Where appreciable amounts of these feedstuffs are not used it may be necessary to include as much as .1 to .15% choline chloride in the ration if trouble from perosis is to be avoided, especially is this true in the case of poult rations.

Gout.

Gout is a chronic ailment that may affect either young or adult birds. It is believed to be due to a disturbance in protein metabolism. Gout is most frequently encountered in birds on high protein rations. Close confinement with accompanying lack of exercise renders birds more susceptible to this disorder.

Two types of gout are recognized, visceral and articular. The visceral form is characterized by poor appetite, sluggishness, anemia, emaciation and diarrhoea. Internal examination of birds, affected with visceral gout reveals kidneys which are often enlarged, pale in color and coated with whitish material (uric acid salts). The heart, liver and other visceral organs may also show similar deposits. Articular gout, on the other hand, is character-

ized by lameness and swelling of the leg, feet and wing joints. The swellings frequently break outwards, discharging white or grayish yellow masses of uric acid salts. Diagnosis of gout is dependent upon microscopic examination of the whitish deposits in acidified water.

Trouble from gout may be prevented by avoiding excess protein feeding and by providing ample opportunity for exercise. The protein requirements of various classes of chickens and turkeys are: growing chickens—20% to 6 weeks, then level gradually decreased to 16% at sexual maturity; growing turkeys—24% to 8 weeks, then level gradually decreased to 18% by 24th week; fattening chickens or turkeys—14%; laying chickens or turkeys—16%.

Treatment of birds affected with gout may be effected by reducing the protein in the diet, supplying an abundance of greenfeed and by providing ample opportunity for exercise.

Obesity.

Obesity is a term used in referring to birds that carry an excess of abdominal fat, and is most frequently encountered in layers and breeders of meaty type. The condition is generally attributed to excessive carbohydrate (grain) feeding. The feeding of large proportions of wheat and barley also seems to cause the condition.

Trouble from obesity may be avoided by feeding layers and breeders, rations of proper nutritive ratio (1:4.5) and rations containing moderate proportions of wheat (30%) and barley (15%). (The ratio of digestible protein to digestible carbohydrate and fat is known as the nutritive ratio.)

MISCELLANEOUS DISORDERS

There are a number of disorders and vices which are not infrequently encountered in poultry and which therefore warrant brief consideration. They include: beak canker, beak necrosis, impaction of the crop, pendulous crop, impaction of the gizzard, bumblefoot, ascites, reproductive disorders, and various forms of cannibalism.

Beak Canker.

Beak canker is a condition that is frequently noted in adult birds. The disorder arises as the result of mechanical injury followed by secondary infection. The most common seat of this trouble is the beak angle. Quite a large swelling may be present. Treatment of beak canker is comparatively simple since it merely entails removing the scab, pressing out the core and pus and swabbing the hole with tincture of iodine.

Beak Necrosis.

This is a disorder that is encountered in chicks fed mash that has been ground too finely. The finely ground feed accumulates in and around the mouth causing inflammation of the tissues and

predisposing to secondary infection. As the condition progresses the beak becomes deformed. Trouble from beak necrosis may be avoided by feeding mashes of coarse texture.

Impaction of the Crop.

This condition, commonly known as crop-bound, is caused by obstruction due to the eating of feathers, straw, long or dead grass or other indigestible material. The symptoms include: loss of appetite, inactivity and general distress. The crop is distended and feels doughy. A sour smell is usually present.

In some cases crop impaction can be corrected by giving an exclusive diet of sour milk, in other cases the condition can be relieved by administering vegetable oil, suspending the bird head down and working the material in the crop out through the mouth. However, in most cases, a minor operation is necessary in order to alleviate the condition. The operation referred to consists of making an incision, about one inch in length, in the upper front surface of the crop (both the outer skin and crop wall are cut through), removing the impacted material through the opening, washing out the crop with warm water to which antiseptic has been added and sewing up the crop wall and outer skin separately with three or four stitches (each stitch is tied separately). Cat gut, silk or cotton thread may be used for sewing. It is not necessary to remove the stitches. For two or three days following the operation only soft food should be fed.

Trouble from crop impaction can be avoided to a considerable extent by keeping mash free of straw and by observing reasonable care in turning birds out in the spring. In the latter connection it is not advisable to turn birds out before grass growth is fairly well under way. Turning birds out before growth has started often results in crop impaction due to birds consuming large quantities of dead grass.

Pendulous Crop.

Pendulous crop is a condition encountered in both chickens and turkeys, though more frequently in the latter. In this disorder the crop is very much enlarged and hangs down in front of the affected bird.

Pendulous crop is an inherited abnormality, though it only appears under certain conditions. The conditions that predispose to pendulous crop are: over-consumption of water, milk or bulky feed, high temperature and low humidity.

Trouble from pendulous crop can be minimized by using as breeders only birds not affected with the abnormality and by avoiding excessive water, milk or bulky feed consumption.

Various methods of treating birds affected with pendulous crop have been tried, but none can be highly recommended. Supporting the crop with cloth or canvas will give temporary relief while tying

off a fold of the crop and allowing it to slough off is a treatment that has been suggested for permanent relief.

Impaction of the Gizzard.

This is a disorder quite frequently noted in birds getting an excessive amount of oat and barley hulls in their feed, or in birds getting a moderate amount of oat and barley hulls in their feed and little or no grit, or in birds that are low in vigour as a result of disease or parasites. Lack of appetite, reluctance to move about and general unthrift are the main symptoms of this disorder. Post-mortem examination of affected birds reveals the presence of excess fibrous material in the gizzard.

Treatment of affected birds may be successful in some instances, and not in others. Recovery is dependent upon the severity of the impaction and the vigour of the individual. Where impaction is due to feeding a ration containing too much oat and barley hulls or too little grit, treatment consists of feeding a ration of lower oat and barley hull content and supplying grit. On the other hand, where impaction is due to some other cause, treatment consists of correcting the primary cause.

Bumblefoot.

The term, bumblefoot, is used in referring to a swelling of the ball of the foot. The condition may arise as a result of foot injury (such as that sustained by birds jumping down from high roosts or nests on to bare floors) and subsequent infection by non-specific infective agents, or may arise as the result of a gouty condition.

Before attempting to treat birds for bumblefoot the predisposing cause or causes must be determined and eliminated. Then the affected birds should be examined and segregated into two groups; those showing the presence of a core, and those not. The birds in the former group should be treated, while those in the latter should be placed back in the flock and examined periodically to see whether recovery or core formation has taken place. Treatment consists of removing the scab, pressing out the core and pus, swabbing the hole with tincture of iodine and bandaging. The treatment should be repeated if deemed necessary. In severe cases poulticing may be necessary to bring relief.

Ascites.

Ascites, also known as dropsy, is a condition often found accompanying enteritis (inflammation of the intestine), peritonitis (inflammation of membrane covering internal organs), fowl tuberculosis, and other diseases affecting the visceral organs. The disorder is not caused by a specific agent.

Ascites is characterized by accumulation of serous fluid in the body cavity and its presence or absence is readily ascertained by palpating the abdomen.

Treatment of birds affected with ascites is useless. When this trouble is detected, a post-mortem should be performed to determine the primary cause in order that appropriate control measures may be adopted.

Reproductive Disorders.

The principal reproductive disorders encountered in poultry are: ruptured yolk, intra-ovulation, impacted oviduct, ruptured oviduct, internal laying, egg bound, prolapsus and cloacitis.

Ruptured Yolk.—This is a condition occasionally encountered in pullets that have been laying at a high rate. Outward evidences of this disorder are sagging of the abdomen and discoloration of the comb. Post-mortem examination shows large quantities of yolk material free in the abdomen. In some cases *Pasteurella avicida,* the fowl cholera organism, has been singled out as the cause, whereas in other instances, excessive production and high roosts have been considered responsible for the trouble.

Intra-ovulation.—Intra-ovulation refers to the ovulation (laying) of unbroken yolks in the body cavity. The abnormality is usually caused by dysfunction of th oviduct (egg tube), though it may also be caused by high roosts or by forcing birds to lay past their productive capacities.

Impacted Oviduct.—Impacted oviduct is another reproductive disorder that is quite commonly encountered. This condition is caused by dysfunction of the oviduct and is characterized by the presence of hardened yolk and albumen (egg white) in the oviduct.

Ruptured Oviduct.—This is a disorder that often develops subsequent to impaction of the oviduct. The break in the wall of the oviduct usually occurs just above the impaction. Birds afflicted with this condition generally lay their eggs into their body cavities.

Internal Laying.—Internal laying refers to the laying of complete eggs in the body cavity. The condition may arise as the result of impaction of the oviduct, rupture of the oviduct or dysfunction of the oviduct. The abdomen of an internal layer is usually quite heavy. Upon palpation, whole or broken eggs may be felt in the abdomen. Internal examination confirms their presence.

Treatment of birds affected with ruptured yolk, intra-ovulation, impacted oviduct, ruptured oviduct or internal laying is in most instances useless. It is much better to attack these troubles from the preventive angle. Keeping birds in good health by proper feeding and management and avoiding excessive production (over 70%) and high roosts (over three feet off the floor) will do much to prevent losses from these disorders.

Egg Bound.—This is a term used in referring to a bird that is unable to expel her egg. The disorder may be due to temporary paralysis of the expelling muscles or to an attempt on the part of the bird to lay too large an egg. The condition can usually be remedied by removing the affected bird to warm quarters, and

applying vaseline or vegetable oil to the tissue around the vent. In instances where this procedures proves unsuccessful, puncturing the egg through the vent with a blunt tool and removing the shell is recommended.

Prolapsus.—Prolapsus, also called blow-out, is a disorder to which very early maturing pullets and pullets just coming into production seem highly susceptible. Birds carrying an excessive amount of abdominal fat and laying at too high a rate, and birds that have been egg bound also seem to be susceptible to this disorder. Prolapsus is characterized by eversion of the cloaca (chamber just inside the vent) and lower oviduct. Birds that have prolapsed, if discovered immediately, can usually be saved by carefully washing and greasing the protruding tissue and pushing it back into place. The oviduct will go back into place more quickly if the affected bird is suspended by the feet for a short time. Affected individuals should be kept separate from the rest of the flock for a week or so. Birds that have prolapsed and that are not discovered soon after, are usually disembowelled and killed by their pen mates. Trouble from prolapsus may be minimized by avoiding too early maturity in pullets, excessive abdominal fat in pullets and hens, and excesses of production (over 70%).

Cloacitis.—Cloacitis, or vent gleet, is generally considered to be a highly infectious venereal disease to which both chickens and turkeys are susceptible. The cause of cloacitis has not as yet been determined. The condition is characterized by swelling, inflammation and ulceration of the cloaca and vent, and by a discharge with an obnoxious odour. The disease is believed to be spread from one female to another by male birds.

Cloacitis is very resistant to treatment. If the trouble is detected early in the course of its development, recovery may be effected by applying a suitable disinfectant repeatedly to the vent. On the other hand, if the disorder is not noticed early in the course of its development, recovery by treatment is uncertain. Among disinfectants that are recommended for treatment of cloacitis are: 3% chromic acid, 3%silver nitrate, 5%carbolic acid, 1:1000 corrosive sublimate, 15% argyrol and 2% carbolic ointment. As a general rule it is advisable to dispose of affected birds as soon as detected.

Cannibalism.

Chickens are decidedly cannibalistic. It seems that once a chicken gets a taste of blood or raw flesh, it must have more of it even at the expense of one or more of its mates.

Toe Picking.—Baby chicks are subject to a form of cannibalism known as toe picking. This vice may be brought on by not having feed down for chicks on arrival at the brooder house, by letting feeding troughs be empty for too long a period, by idleness, by overcrowding, by overheating, by having too much light in the brooder house, or by permitting sun spots in the brooder house. Where toe

picking has broken out, the predisposing cause should be determined and corrected. Next the windows should be given a coat of red show card paint and all white light should be excluded. The red light prevents, to a considerable extent, the chicks from seeing the blood on the toes of injured chicks, and so the habit is curbed.

Tail Picking.—This is another type of cannibalism that is encountered in growing chickens. It often follows toe picking. It may also occur in older chickens. Among the factors that predispose to tail picking are: idleness, overcrowding, overheating, too close confinement and letting feed troughs be empty for too long a period. Any factor that causes the chicks to become restless may be considered as predisposing to tail picking. Where tail picking has broken out the predisposing cause should be determined and corrected. Next, all the tail-picked birds, the "pickers" and a number of chicks selected at random should be smeared on the tail and wings with some anti-picking material such as pine tar or a mixture of 4 ounces of vaseline plus ¼ ounce of carmine plus ½ ounce of aloes. If th chicks are not running outside and weather conditions are such that they might be, every effort should be made to get them outside. Giving the chicks free range, and avoiding overcrowding will do much towards bringing this vice under control.

Two further aids to bringing an outbreak of tail picking under control are the hanging of red buttons from the ceiling at a height such that the chicks will have to jump to pick at them, and the feeding of salt in the drinking water. In the latter connection it has beeen found that one tablespoon of salt should be added to each gallon of drinking water and that the birds should have access to the salt water on two or three forenoons, each three days apart.

Feather Picking.—A further cannibalistic habit is feather picking. This habit may be encountered either in developing birds or layers, though it is most frequently noted in the latter. When feather picking is present in a flock, birds may be seen to go about picking out feathers from the backs or from around the vents of their pen mates. If the vice has not been recognized at an early stage the backs and abdoments of most of the birds in the flock may be completely devoid of feathers. Feather picking is believed to be brought on by lack of one or more nutrients in the diet. The nutrient or nutrients responsible for the prevention of this habit have been shown to be present in oats. In this regard it has been shown that the inclusion of a liberal amount (30% of the ration) of oats in the diet of poultry will prevent the appearance of feather picking. An outbreak of feather picking may often be brought under control by including oats in the diet at the proper level and giving salt in the drinking water (see Tail Picking). Sometimes it is difficult to break the habit especially when the birds are kept confined.

Vent Picking.—Another vice which chickens frequently acquire is vent picking. Vent picking often breaks out following the disembowelling of two or three birds that have prolapsed. It may also accompany feather picking or it may have its origin with birds picking others that are laying. Vent picking is a vice that must be given prompt attention, otherwise serious losses may be sustained. A few offenders in a flock can teach the vice to the entire flock is a very short time, and in the course of a few hours many birds will have been disembowelled, and practically every bird in the flock will be bleeding at the vent. Offending birds should be detected and should either be isolated from the main flock for two to three weeks to break them of the habit or have anti-picking devices (obtainable from the larger poultry supply houses) placed on their beaks to prevent them from picking. Birds that have already been picked should have their vents smeared with an anti-picking material such as one of the materials referred to under Tail Picking. Darkening the nests, including a liberal amount of oats in the diet and giving salt in the drinking water (see Tail Picking) will also aid in bringing this habit under control.

EXTERNAL PARASITES

External parasites affect poultry adversely. The irritation caused by their movement, gnawing, and sucking, leads to restlessness in the bird. Restlessness in turn tends to throw the bird out of physiological balance. When this occurs, digestion and assimilation are slowed up. As a result, feed consumption drops. In young birds the decrease in feed consumption is paralleled by a marked decrease in the growth rate. Severe infestation may even end in death. In adult birds the decrease in feed consumption is coupled with a decrease in body weight and a falling off in egg production. In general, external parasites tend to bring about a state of unthriftiness, lowered vitality and lowered resistance to disease. There are two main types of external parasites that affect poultry, namely, lice and mites.

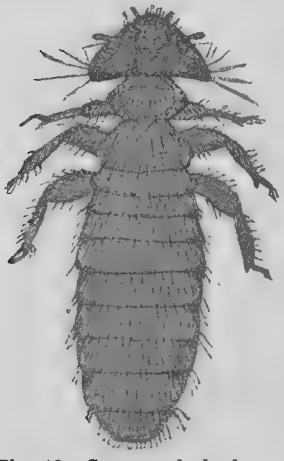

Fig. 16.—Common body louse. (After Osborne.)

Lice.

Lice are small yellowish or grayish flattened insects (see Fig. 16). Their bodies are divided into three regions: head, thorax and abdomen. They have three pairs of legs and their mouths are equipped for biting and cutting. They do not suck blood, but feed on bits of feather, scales from the skin, and dried blood.

There are about fifty species of lice that infest domestic poultry. The common body

louse (*Menopon stramineum*) found principally in the region of the vent; the shaft louse (*Menopon gallinae*) located along the shafts of the feathers; the head louse (*Lipeurus heterographus*) found in the head and neck region, and the wing louse (*Lipeurus viriabilis*), detected along the primary feathers, are amongst the more important chicken lice, while the large turkey louse (*Goniodes meleagridis*) is the most important turkey louse.

All species complete theilr life cycle on the bird. For the sake of an example let us consider the common body louse. In this species eggs are deposited in large masses at the base of feathers, particularly in the region of the vent. These eggs, under favourable conditions, hatch in from five to seven days to produce nymphs. The nymphs, after several moults, give rise to adults. The whole process from egg to adult takes from two to three weeks.

There are several satisfactory methods of treating for lice.

Birds may be freed of lice by dusting with a suitable material. Sodium fluoride and sodium fluosilicate are amongst the most effective and inexpensive materials known. Pinches of powder are applied to various parts of the bird's body, below the vent, in the thigh fluff, on the breast, under the wings, over the back and in the neck. One pound of powder used in this manner will treat about one hundred birds. Sulphur may also be used, but it has not been found as effective as either of the previously mentioned powders. When used, it is applied at the rate of six pounds per hundred birds. A home-made powder that has proven very efficient is prepared by adding to one part of crude carbolic acid and three parts of gasoline, sufficient plaster of paris to take up the moisture. The liquid and the plaster are mixed until a dry powder is left. The prepared powder is dusted into the feathers in a manner similar to that indicated for sodium fluoride and sodium fluosilicate. It is claimed that the cost of treating birds with this preparation is much lower than with sodium fluoride. Regardless of the dusting material used, it is necessary to give a second treatment ten days after the first in order to kill any lice that may have hatched from eggs that were on the birds at the time of the first dusting.

Dipping of birds in warm water containing some material effective in destroying lice may be practised during warm weather. Sodium fluoride and sodium fluosilicate are the chief substances used in dipping solutions. One ounce of commercial powder or two-thirds of an ounce of chemically pure powder is added to each gallon of water. One pound of powder makes sufficient solution for dipping upwards of three hundred birds. In practice the birds are immersed, with the exception of the head, for twenty to thirty seconds. The feathers are ruffled so as to permit the dip to penetrate to the skin. Care should be taken to see that the dipped birds have ample time in which to dry off before night. A second treatment ten days after the first is essential.

Mild greases such as lard and carbolated vaseline smeared on the top and back of the head may be used effectively in controlling head lice in young birds. Mercuric ointment, commonly known as blue ointment, diluted with vaseline 1:1 or a mixture of one part lard and one part sodium fluoride, have been found effective in combatting lice in older birds. A small quantity of the grease is applied just below the vent and under the wings. A second treatment is seldom required as the effect is lasting.

Fumigating with nicotine sulphate (Black Leaf Forty) is about the easiest treatment yet discovered. It is not as effective as some of the aforementioned treatments. The method consists of applying nicotine sulphate to the tops of the perches at the rate of eight ounces per 100 linear feet just before roosting time. The heat of the fowls' bodies causes the volatile nicotine sulphate to vaporize, the fumes penetrate the feathers of the birds and kill the lice. The roosts should be scraped free of organic material before the nicotine sulphate is applied. Care has to be taken to see that all birds roost and that no drafts are present, otherwise the treatment will not be successful. This treatment should only be used when the room temperature is over 55°F, and to be effective must be repeated in ten days time. Another method of using nicotine sulphate (one which costs less but involves more labour) is to apply it a drop at a time, by means of an oil can, directly to the bodies of the birds, under the wings and in the fluff around the vent. No second treatment is necessary in this case. .

The treatment of birds for lice should be followed by a thorough cleaning of the houses and premises. This is necessary, since the houses and runs are contaminated with lice and lice-egg-laden feathers dropped from infested birds. If these are not destroyed, reinfestation will result.

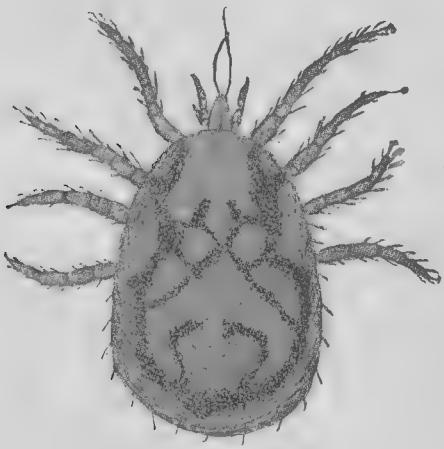

Fig. 17.—Common chicken mite. (After Hutyra and Marck.)

For preventive measures for lice, see Mites.

Mites.

There are about twenty species of mites that infest domestic poultry. Of these, only four are sufficiently injurious to be of economic importance. They are the common chicken mite (*Dermanyssus gallinae*), the northern fowl mite (*Liponyssus silviarum*), the depluming mite (*Cnemidocoptes gallinae*), and the scaly leg mite (*Cnemidocoptes mutans*).

Mites are oval bodied creatures (see Fig. 17). Their

bodies do not show head, thorax and abdominal divisions characteristic of lice. Their mouths are equipped for sucking blood.

Some species of mites complete their life cycle on the bird while others do not. Generally speaking, the time needed to complete the life cycle is short; the average time required from egg to adult, under favourable conditions, being seven or eight days.

The Common Chicken Mite.—The common chicken mite, also referred to as the red mite, because of its red appearance when gorged with blood, is gray in color. This mite does not stay on the fowl all the time. It is found on the bird only at night (in cases where infestation is exceedingly heavy it is sometimes found on the bird during the day). During the day it hides in cracks and crevices. Its presence may be determined by examining the ends and undersides of the roosts at the points of support, and by careful examination of any cracks or crevices in the roosts or roost supports. "Salt-and-pepper markings" (the excrement and sloughed-off skins of mites) under or on the roosts are also an indication of the presence of red mites. Examining a representative group of birds after dark is another way in which the presence or absence of this mite may be determined.

If evidence of infestation is seen, measures of control should be taken at once. The house and house equipment should be cleaned. Roosts, dropping boards and nest boxes should be thoroughly sprayed. Among the most effective materials that may be used for spraying are: undiluted creosote, anthracene oil, crude petroleum, crank case oil, coal tar dips (10% in water), and 3% miscible tree spray. Following cleaning and spraying, the house should be whitewashed so that every crack is filled.

The Northern Fowl Mite.—This parasite somewhat resembles the common chicken mite in appearance. It differs, however, in habit, in that it completes its life cycle on the bird and accordingly its presence or absence may be ascertained by examining the fowl at any time. Northern fowl mites tend to congregate in certain areas for feeding. Often the injury inflicted in these areas is so severe that large scabs form.

There are at least three satisfactory methods of treating for this mite, namely, fumigating with nicotine sulphate (Black Leaf Forty), greasing with a mixture of one part napthalene to two parts vaseline, or, during warm weather, by dipping the birds in a solution of two ounces of sulphur plus once ounce of soap in one gallon of warm water. Fumigation with nicotine sulphate is carried out in a manner similar to that outlined for lice (see Lice), except that three applications at intervals of three days are necessary in this instance. The method followed in greasing and dipping is the same as that described under Lice.

The Scaly Leg Mite.—This mite burrows under the scales of the shanks, causing the condition known as scaly-leg (see Fig. 18). The scaly leg mite completes its life cycle on the bird.

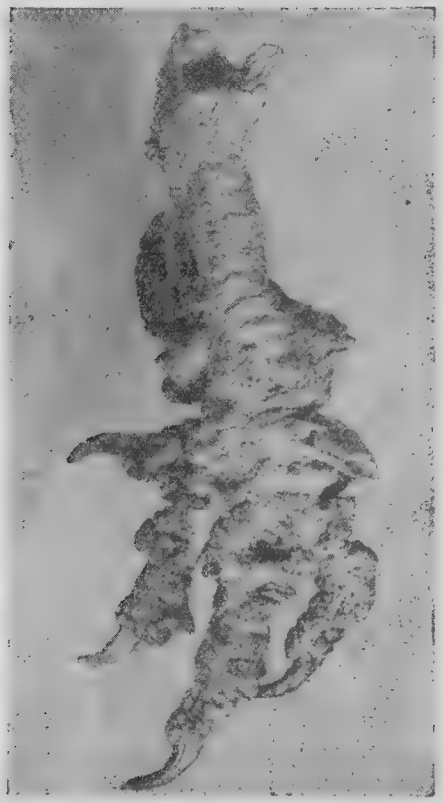

Fig. 18.—A bad case of scaly-leg. This condition is caused by the scaly leg mite. (After Kansas State Agricultural College.)

Treatment may be effected by applying to the shanks an ointment of 1 part of oil of caraway to 5 parts of vaseline; or by dipping the shanks in petroleum, crank case oil, kerosene, a mixture of 1 part kerosene to 2 parts raw linseed oil; or in a mixture of 1 part crude petroleum to 1 part raw linseed oil. In connection with the ointment treatment, before applying the salve it is advisable to soak the shanks in warm soap suds until the scales are loose. Two applications at three or four day intervals are necessary. Where the dipping-of-shanks method of treatment is followed, care should be taken to see that the dip does not get on the skin of the bird, otherwise burning will result.

The Depluming Mite.—The depluming mite burrows into the skin at the base of the feathers, causing an intense itching, which causes the bird to pick out its feathers. As a result, large areas on the bird may be devoid of feathers.

Treatment may be accomplished by applying to the affected areas either oil of caraway ointment (see above), or sulphur ointment (1 part flowers of sulphur to 4 parts lard or vaseline), or, during warm weather, by dipping in a solution of two ounces of sulphur plus one ounce of soap to one gallon of warm water. At least two applications of ointment should be given. Where dipping is resorted to, the birds must be thoroughly soaked for the treatment to be effective.

While definite methods of treatment for lice and mites have been outlined, it should be realized that cleanliness and proper housing constitute the most effective means of controlling external

parasites. Houses should be thoroughly cleaned, sprayed and whitewashed as least once a year, better twice; litter and nesting material should be changed at least every two weeks; birds should be dusted at least twice a year; dust baths, consisting of ashes plus one of the previously mentioned dusting materials, should be provided and houses and equipment should be so constructed as to provide for a minimum of cracks in which mites might hide and breed.

INTERNAL PARASITES

There are two main types of worms that affect poultry, namely, roundworms (*Nematodes*) and tapeworms (*Cestodes*). Roundworms are readily distinguished from tapeworms by morphology, the former being round and unsegmented, the latter flat and segmented. Both types are usually white in colour and vary in length according to species. The large intestinal roundworm (*Ascaridia lineata*), the caecal worm (*Heterakis gallinae*), and the gapeworm (*Syngamus trachea*) are among the more important roundworms that infest the fowl. Several species of tapeworm are known to infest the fowl.

The Large Intestinal Roundworm.

This worm (see Fig. 19) inhabits the small intestine. It is white in colour and varies in length from one to three inches. Its life cycle is direct (see Fig. 20). Non-infective eggs (non-infective to the fowl) are laid in the intestine by the adult female worm. These eggs pass out of the body along with the faeces. Under suitable conditions of moisture and heat, these non-infective eggs undergo development (embryonation) to give rise to infective eggs. Upon being ingested by a fowl these infective eggs pass down into the small intestine where they liberate larval worms. The larval worms so released enter the intestinal wall where they undergo further development to produce normal worms which, after passing out into the intestine, grow to maturity and lay their complement of non-infective eggs.

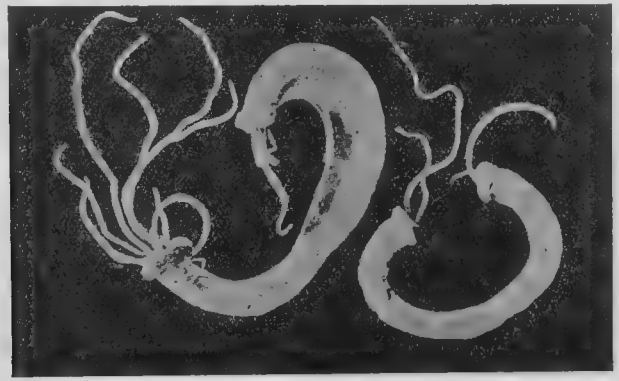

Fig. 19.—Section of intestine showing large intestinal roundworms protruding. (After Bradshaw.)

Old birds are less susceptible to large intestinal roundworm infestation than young birds. This difference is due to the development of a so-called "age immunity" as fowls grow older. Birds on an inadequate diet are more susceptible to intestinal roundworm infestation than are birds receiving a properly balanced diet.

The principal symptoms of large roundworm infestation in growing fowls are: paleness of head and shanks, loss of appetite, stunted growth, emaciation, inactivity, and droopiness. In advanced cases lameness or paralysis is often noted. In mature fowls decreased egg production, loss of weight and general unthrift should be regarded as indications of roundworm infestation.

While the above symptoms are suggestive of the presence of roundworms, they do not constitute conclusive evidence. The presence or absence of roundworm infestation is best ascertained by examining the intestinal contents of suspected birds.

On farms where infestation prevailed in the previous season, ground over which the birds ranged constitutes the main reservoir of infection. This is due to the fact that worm eggs are able to persist in th soil in a viable state from one season to the next. On farms where current infestation exists, the dropping boards (especially if not cleaned daily), litter, contaminated feed and water, and the ground over which the birds range, comprise the principal sources from which healthy fowls contract infection.

Worm infestation has a decided detrimental effect on vigour. Worm infested birds are much more susceptible to disease (e.g.,

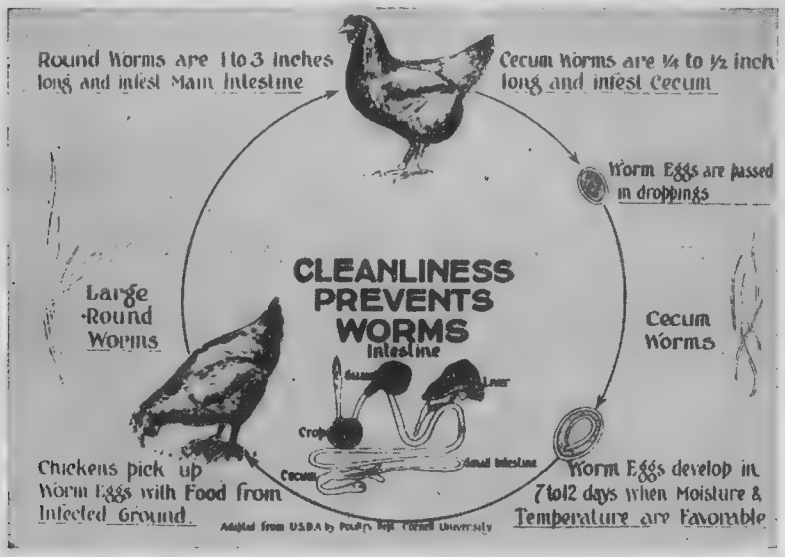
Round Worms are 1 to 3 inches long and infest Main Intestine
Cecum Worms are ¼ to ½ inch long and infest Cecum
Worm Eggs are passed in droppings
CLEANLINESS PREVENTS WORMS
Intestine
Large Round Worms
Cecum Worms
Crop
Liver
Cecum
Chickens pick up Worm Eggs with Food from Infected Ground.
Worm Eggs develop in 7 to 12 days when Moisture & Temperature are Favorable
Adapted from U.S.D.A. by Poultry Dept. Cornell University

Fig. 20.—Life cycle of large intestinal roundworm and of caecal worm. (After Cornell University.)

colds) than healthy birds. Mortality from roundworm infestation varies greatly. In general, losses in young stock are more serious than in adult birds.

Trouble from roundworms may be avoided by observing:

(1) Sanitation on the range.

(a) Using poultry ranges one year in two; or better, one year in three (to ensure freedom from worm eggs).

(b) Avoiding damp spots on the range (such spots provide an ideal environment for the embryonation of worm eggs). All low damp spots should be filled in or fenced off. Unused drinking water should be disposed of in such a manner as not to leave puddles on the range. Water fountains should be placed up on wire stands to prevent birds having access to the damp ground usually present about them.

(2) Sanitation in the brooder house.

(a) Cleaning out the brooder house thoroughly before use (to destroy worm eggs that may be present). Iodine Suspensoid (Merck & Co.) or lye water (1 pound lye to 5 gallons water) should be used for disinfecting the floor.

(b) Using wire under the roosts (to prevent the birds from coming in contact with their faeces).

(c) Cleaning the litter and droppings out at regular intervals (to lessen the chances of worm spread).

(3) Sanitation in the laying house.

(a) Cleaning out the laying house thoroughly prior to housing pullets (to destroy worm eggs that may be present). See recommendations above for disinfection.

(b) Cleaning the dropping boards daily or using wire netting under the roosts (to preevent the birds from coming in contact with their faeces).

(c) Cleaning the litter out at regular intervals (to lessen the chances of worm spread).

(4) Sanitation in feeding.

Protecting feed and water against contamination with droppings (to avoid spreading infection). All feeders and waterers should be provided with reels to prevent birds getting into them. Landing boards in front of feeders and waterers should be sufficiently far away from same to prevent birds from soiling feed and water with droppings.

(5) Sanitation in management.

(a) Rearing young stock separate from old stock (the latter frequently harbour worms, hence must be considered as a potential source of infestation).

(b) Taking precaution against mechanical transmission of infection from old stock to the young stock. Rubbers should be put on at the time of entering the young stock range.

(c) Being careful not to bring infection on the farm. Imported birds should be treated for worms if infestation is suspected. Visitors should be required to disinfect their shoes before being shown around the farm.

Treatment of birds infested with roundworms may be effected by administering a vermifuge. Several good vermifuges are obtainable on the market, e.g., Iodine Vermicide (Merck & Co.), Nema Capsules (Parke Davis & Co.), Nico-gel Pills (Lederle Laboratories Inc.), and Black Leaf Worming Pellets. Two chemicals that are recommended widely for treatment of roundworms are tetrachlorethylene and carbon tetrachloride. One cubic centimeter of either constitutes a dose for an adult bird, while 0.5 c.c. is sufficient for a growing bird. The following procedure is recommended when either of these two chemicals is used:

(1) Treat affected birds in the morning before they are permitted to feed.
(2) Administer the proper amount of chemical into the crop of each bird, either in capsule form or by means of a glass or rubber tube.
(3) Give Epsom salts in the drinking water at the rate of one pound per hundred birds.
(4) Supply regular feed two hours after treatment.
(5) Confine birds to a house for three days following vermifuge administration.
(6) Clean the house out thoroughly on the third day, using Iodine Suspensoid or lye water for disinfecting.

Following the treatment every effort should be made to avoid reinfestation. Under no circumstances should the treated birds be permitted to range over ground on which they previously ranged. Young stock in colony houses should be moved on to a clean piece of range, while old birds in permanent houses should be confined until such time as their yards have been cleaned up.

Worm-egg-infested ground is best cleaned up by ploughing and cultivating frequently. One summer's working and a year's rest will clean up a worm-infested range.

The Caecal Worm.

These small white worms (¼ to ¾ inches long) inhabit the blind guts or caeca of the birds. The life cycle of this worm is similar to that of the large intestinal roundworm (see Fig. 20).

Most classes of poultry are susceptible to caecal worm infestation. Birds in a state of malnutrition (e.g. deficient in vitamin A) are more susceptible to caecal worms than those in a state of proper nutrition.

The symptoms, sources of infection, and effects of caecal worm infestation are similar to those referred to under Large Intestinal Roundworm. Post-mortem examination of infested birds reveals large numbers of worms toward the tips of the caeca.

It should be pointed out that the caecal worm is dangerous in the poultry yard, not only because it is capable of infesting birds and causing trouble, but also because of the part it plays in the transmission of blackhead (see Blackhead).

Preventive measures for caecal worms are the same as for large intestinal roundworms (see Large Intestinal Roundworm).

Treatment of infested birds is somewhat difficult as the caecal worm inhabits a rather inaccessible part of the intestine. Administering 2%, by weight, of freshly pulverized tobacco (containing not less than 1½% nicotine) in the mash for four to six weeks, or giving birds over three pounds in weight a rectal injection of 2/3 of a fluid ounce (birds under three pounds in weight 1/3 of a fluid ounce) of a mixture of 1 fluid drachm of oil of chenopodium (oil of American wormseed) and 6 fluid ounces of vegetable oil, are two common methods of treating for caecal worms.

In connection with the former treatment, the mash and tobacco dust mixture should be made up frequently in small amounts rather than in one large lot. This is advisable because the tobacco loses its nicotine rapidly on exposure to air. In the latter treatment, an enema syringe with a small blunt nozzle is used for injecting the oil of chenopodium-vegetable oil mixture.

A third method for treating for caecal worms and one that is highly effective consists of administering the drug phenothiasine at the rate of one pound per thousand chickens or growing turkeys, or at the rate of two pounds per thousand adult turkeys. The drug may be given either in capsule form or may be fed in the mash.

Regardless of the treatment used, reinfestation must be avoided by following a plan similar to that given under Large Intestinal Roundworm.

The Gapeworm.

Gapeworms are small Y-shaped worms of reddish brown colour. The short part of the "Y" is the male and the long part the female. These worms attach themselves to the mucous membrane of the trachea (windpipe) and suck their food (blood) therefrom. The life cycle of this worm is similar to that of the large intestinal roundworm (see Fig. 20).

Both chickens and turkeys are highly susceptible to gapeworm infestation. Heavy losses may be experienced where chickens, especially young chickens, are infested with these worms. Turkeys, however, may harbour numerous worms without showing bad effects.

The chief symptoms noted in gapeworm infestation are gasping for breath and inactivity. Death occurs through suffocation. Post-

mortem examination reveals the presence of reddish brown Y-shaped worms in the windpipe.

Sources of infection for gapeworms are similar to those referred to under Large Intestinal Roundworm.

Preventive measures for gapeworms are the same as for large intestinal roundworms (see Large Intestinal Roundworm); with one addition, namely, that chickens should be reared separate from turkeys. This is advisable since turkeys are potential carriers of gapeworms.

A treatment that has proven successful in removing gapeworms consists of pipetting 1/3 to 3 minims of a mixture of equal parts garlic oil or allyl sulphide and linseed oil into the windpipe of affected birds. The drug causes the parasites to let go their hold and the birds cough them up. An alternative treatment that is highly recommended consists of exposing affected birds to air containing barium antimonyl tartrate dust. The birds are exposed to the chemical in a closed container, the size of the dose being determined by the cubic capacity of the container used. One ounce of barium antimonyl tartrate is sufficient for a box having a capacity of 8 cubic feet. Infested birds are placed in the box in sufficient numbers to occupy about two-thirds of the floor space. The barium antimonyl tartrate is introduced with a dust gun into the box through a hole in the top. The chemical should be introduced gradually and the box should be agitated from time to time. The treatment should take about 15 minutes.

Reinfestation should be guarded against by following a plan similar to that outlined under Large Intestinal Roundworm.

Tapeworms.

As previously stated, tapeworms are flat, segmented worms of white colour (see Fig. 21). The species that infest poultry vary in size from those that cannot be seen by the unaided eye to worms of nine inches in length. The life cycle of the tapeworm is indirect, i.e., one phase of it is spent inside the body of a secondary or intermediate host (see Fig. 22). Non-infective eggs (non-infective to the fowl) are passed in the droppings of infested birds. Then these non-infective eggs are ingested by suitable secondary or intermdiate hosts (fly larvae, flies, snails, beetles, grasshoppers, earthworms, etc.). While inside the intermediate host, they undergo development to produce infective larvae (cysticerci). Next the intermediate host containing the infective larva is eaten by the fowl. During the course of digestion, the head (scolex) within the cyst is released and attaches

Fig. 21. — Section of intestine showing tapeworms attached. (After Ackert.)

itself to the wall of the upper intestine by means of hooks and suckers. During the course of developement that ensues, new segments (proglottids) are formed in the region behind the head (strobilus). This development continues until the tapeworm has grown its characteristic length, at which time the oldest proglottids (which are filled with non-infective eggs) detach, to be later passed out of the body of the fowl with the faeces.

Chickens and turkeys are both susceptible to tapeworms.

The symptoms of tapeworm infestation are similar to those enumerated under Large Intestinal Roundworm. In heavy tapeworm infestations segments can often be observed in the droppings.

Post-mortem examination of affected birds reveals an inflamed upper intestine and the presence in the intestine of segmented, flat, white worms. Of course, where microscopic species of tapeworms are concerned, it is impossible to see the worms without the aid of a microscope. One species of tapeworm (*Raillietina echinobothrida*) produces small nodules in the walls of the intestine. These nodules should not be mistaken for tuberculosis nodules.

The damage done by a few tapeworms in mature birds is not very great, but a heavy infestation, especially in young birds, may have disastrous results. It should, however, be pointed out that tapeworm infestation, regardless of the severity of the infestation, like roundworm infestation tends to undermine the bird's vitality and so predispose to secondary infection. This in itself

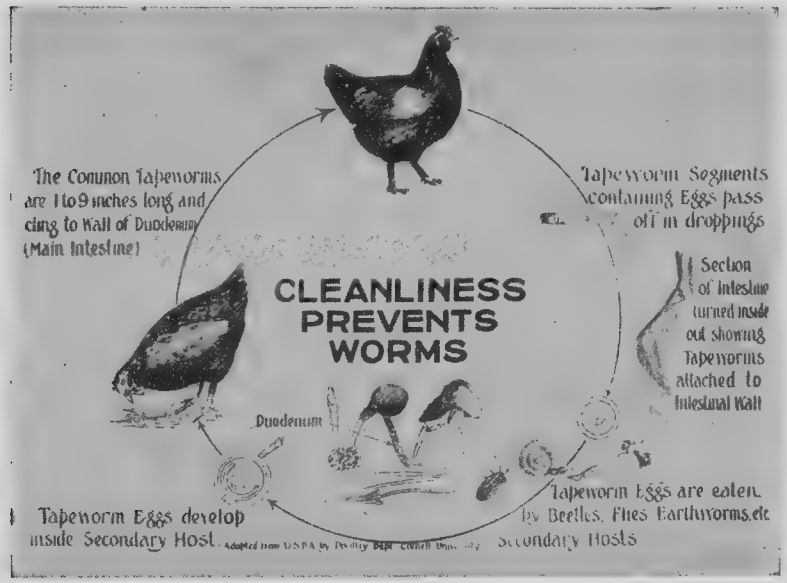
The Common Tapeworms are 1 to 9 inches long and cling to Wall of Duodenum (Main Intestine)
Tapeworm Segments containing Eggs pass off in droppings
Section of Intestine turned inside out showing Tapeworms attached to Intestinal Wall
CLEANLINESS PREVENTS WORMS
Duodenum
Tapeworm Eggs develop inside Secondary Host
Tapeworm Eggs are eaten by Beetles, Flies Earthworms etc Secondary Hosts

Fig. 22.—Life cycle of poultry tapeworm. (After Cornell University.)

constitutes sufficient reason for taking precautions to control the tapeworm menace.

The preventive measures listed under Large Intestinal Roundworm are applicable to tapeworms. In addition any measure that will lead to a reduction in the number of intermediate hosts on the poultry farm (such as the removal of all old boards and debris, etc., which provide a breeding place for intermediate hosts) or to prevention of intermediate hosts from coming in contact with worm egg infected droppings (such as by frequent removal of droppings and litter) will prove of value in combatting the tapeworm problem.

Treatment of birds infested with tapeworms is very difficult. No chemical is known which, when administered to an infested bird, will cause the expulsion of all tapeworms intact. Vermifuges are known that will destroy all the segments, but none are known that will destroy all of the heads. The latter remain attached to the intestinal wall and develop a new lot of segments. Hence, administering a vermifuge for tapeworms merely results in a temporary cessation of tapeworm activity. To be of any material value a tapeworm vermifuge should be administered two or three times at three week intervals, and reinfestation should be guarded against. Iodine Vermicide (Merck & Co.) and Rota-caps (Dr. Salisbury's) are considered amongst the best tapeworm vermifuges available. For details of administration the reader is referred to the manufacturers' instructions.